1.
April 1, 2019

Conard Metcalf
Simmons Hanly Conroy
One Court Street
Alton, IL 62002

Re: Karen Cahoon, As Executrix Of The Estate Of Grace Webster, Deceased, Plaintiffs, v. Edward Orton, Jr. Ceramic Foundation, Metropolitan Life Insurance Company, Defendants.

Dear Mr. Metcalf,

I have been retained by Simmons Hanly Conroy to provide industrial hygiene opinions in the above captioned matter. The following report represents a synopsis of and basis for my opinions with regard to *Grace Webster*'s exposure to asbestos through her used of Orton Pyrometric Cones packed in vermiculite. I have reviewed the materials provided to me pertaining to *Grace Webster*. My report regarding her exposure to asbestos follows. My opinions are based on a more probable than not basis and are a result of my education, training, experience, and my review of the literature and documents cited in this report. I reserve the right to alter my opinions based on additional discovery information.

2. Introduction

I am Terry M. Spear. I reside at 110 South Hauser, Anaconda, Montana.

3. Qualifications General

I hold a Ph.D. in industrial hygiene. I am a Professor Emeritus of industrial hygiene at Montana Tech of the University of Montana in Butte where I formerly taught courses in industrial hygiene and served as the head of the Safety, Health, and Industrial Hygiene Department. I have served as a professional industrial hygienist in industry and academia and have evaluated hazards relating to asbestos in both of those settings. I have been conducting research relating to aerosols for 27 years. I have 20 peer-reviewed publications on aerosols, including seven publications on the subject of asbestos. My Curriculum Vitae is Appendix #1 to this expert report. My billing fee for document review and testimony preparation, deposition testimony, and trial testimony is $300, $400, and $500 per hour, respectively.

4. Qualifications

I have experience in the field of industrial hygiene pertaining to asbestos and have worked on industrial hygiene issues relating to the W.R. Grace mine near Libby, Montana for about 20 years. I have extensively studied and provided testimony regarding asbestos exposure in and around Libby, including during mining activities, railroad activities, and during the use of vermiculite products contaminated with asbestos. I have also testified in cases involving asbestos exposure in Libby at the former vermiculite mine, at the Libby Lumbermill, and at various workplaces in the Libby area. I have visited Libby on many occasions, and have visited the former vermiculite mine and the Libby Lumbermill. I have interviewed scores of former lumber mill and mine workers, as well as citizens of Libby. I have reviewed thousands of records from W.R. Grace, Zonolite, and governmental agencies including State of Montana

agency records, records of third parties, relevant industrial hygiene literature, and applicable statutes, regulations and court decisions interpreting statutes and regulations. All of these materials are the type regularly relied upon by professional industrial hygienists in the performance of their profession. I served three years as the Technical Advisor for the Libby Area Technical Advisory Group (LATAG). I am co-author on seven peer-reviewed publications pertaining to Libby amphibole asbestos, and am still involved in research in the Libby area.

5. Grace Webster History

Grace "Holly" Webster was born October 16, 1929 in Pasquotank, NC. She passed away on July 8, 2016. Her death is attributed to malignant mesothelioma. . The following exposure and work history is a result of the investigation into Holly's toxic exposures to asbestos. This summary is current as of, January 29, 2019. The investigation into potential asbestos exposures is continuing.

Ceramics Teacher (1968-Early 2000's)

As far back as her daughter, Karen Cahoon, can remember, Holly Webster taught ceramics. She taught between four and six classes a week for the College of the Albamarle. In approximately 1968, Holly and her then husband, William Paul Bunnell, bought a home in Elizabeth City, NC. Upon buying the home, they converted the one car garage into a ceramics studio where Holly taught for the local college and for others in the community looking to take classes.

The garage itself was separated into two parts: one area where tables were setup for teaching and another area that housed two kilns for firing the ceramics. The area that housed the kilns was approximately five feet by ten feet, with walls that ran to the ceiling and the kilns were located in the upper right-hand corner. There was a workbench where the Orton Pyrometric Cones were stored and on the left were shelves where students would place their ceramic pieces to be fired. Karen Cahoon testified that Holly "would move from the shelf to the kilns, to the shelf, to the kilns, and it was right there in that small space." Karen Cahoon testified that the ventilation was poor in the back room and there was no ventilation system. There were no windows.

As a regular part of her ceramics work, Holly used Orton Pyrometric Cones. Karen Cahoon would go with her mother from time to time to purchase some ceramic supplies at Stu-doodle in Newland, to where Holly would make weekly or biweekly trips to by Orton Pyrometric Cones. These cones were used to help fire the kilns at the proper temperature. There were generally 50 Orton Cones in each box. The Orton Cones were packaged in vermiculite. In order to get a cone out of the box to use in the kilns, Holly would have to dig around in these boxes to locate and extract a cone. Alternatively, Holly would often dump the boxes of cones and associated packaging materials onto a tray or into a larger bowl to make it easier to sift through. Fishing around in the vermiculite materials and dumping the materials into a larger surface both generated visible dust. As the Pyrometric Cones were retrieved, Holly would often use her hand to brush excess vermiculite material into the trash or onto the floor. Any vermiculite packaging material that had been brushed onto the floor was cleaned up by Holly using a small broom

and dust pan, sand then dumped into a trash can. The process of pouring that material into the trash can created visible dust. The vermiculite packaging materials that made it to the floor would often remain on the floor for days before being swept up. When the vermiculite was on the floor, people would walk on the vermiculite packing and the vermiculite debris would be tracked around the garage/studio. When asked how often she observed her mother in those dusty conditions, Karen Cahoon testified “I'm not sure how to put a number on that because if she was firing, which seemed to be almost all the time, then her being in those conditions goes hand and hand with that. So I don't know how to put a number on that.” If Holly wasn't in that classroom teaching, she was back there firing the kilns.

At all times that Holly was using Orton Pyrometric Cones, it would have been in this confined kiln area at the back of her ceramics studio. Only Holly and her daughter Karen were allowed to fire the kilns, so Holly was typically using Orton Cones in the manner described on a daily basis.

In approximately 1992, Holly moved from the Elizabeth City home to another house adjacent to her daughter’s. Between the two homes was a large barn that was partitioned into several different rooms. Holly continued her ceramics teachings at this location until the early 2000’s. Again, there was a room dedicated to teaching and another area where the kilns were located and Orton Pyrometric Cones were used. The firing area was approximately the size of a one car garage and had one window on each side. The other two sides were floor to ceiling walls. Other than a door on each side of the room to pass through, there was no ventilation. Holly continued her work in substantially the same manner, using Orton Pyrometric Cones daily to fire the kilns.

It was in this firing room after her passing that Holly’s daughter, Karen, found 18 boxes of Orton Pyrometric Cones. These cones were sent to MVA Scientific Consultants. The MVA testing was be furnished separately. At this time Karen also found some of the cones were actually packaged in Styrofoam. Karen never remembered in your experience removing cones from Styrofoam, and she only remembers the cones packaged in vermiculite. On page 6 of Exhibit 3 Karen read the warning label from the box of Orton cones: "Warning. Keep out of reach of children! Cone numbers 022, 021 and 020, contain lead compounds which may be harmful if swallowed." Additional information read “Important notice to purchaser. Orton Pyrometric Cones will be replaced if defective in manufacture, labeling or packaging. Except for such replacement the sale of Orton Pyrometric Cones is without other warranty or liability. User shall determine the suitability of this product for the intended use, and assume all risk and liability in connection therewith." Those are the same warnings and notices that Karen remembers. Throughout that entire period of time from approximately the late 1960s to early 1970s until her mother ceased performing ceramics work in sometime in the late 1990s or 2000, Karen never saw any warnings about the hazards of asbestos on any of the packaging of Orton cones.

Beyond her work as a ceramics teacher, there is no evidence that Holly had any other exposures to asbestos.

6. Orton Company Sold Vermiculite Containing Libby Amphibole Asbestos (LAA)

The Edward Orton Jr. Ceramic Foundation was established in 1932 to continue the operation of the Standard Pyrometric Cone Company for the benefit of the ceramic arts and industry. Orton is the longest continuous manufacturer of pyrometric cones in the world, and ships to over 70 countries worldwide (Exhibit 1, History of Orton). Orton began manufacturing pyrometric cones in 1896. As of 2007, Orton was selling over 12 million cones per year; this number had decreased from its peak sales in the 1970's and 1980's (Exhibit 2, Deposition of Orton's Corporate Designee, Gary Childress, 11/28/07 at 20, 115).

Prior to 1963, Orton packaged its pyrometric cones in sawdust (Exhibit 3, Deposition of Orton's Corporate Designee, Dale Fronk, 5/8/08 at 11-12). In 1963, Orton made the decision to cease packing these cones in sawdust due difficulties in obtaining it the quantities Orton required. Id. At that time, Orton looked at six to eight alternatives to sawdust, and subsequently decided to begin using vermiculite from Libby, Montana for its packaging material. Id. Other than Libby vermiculite, other alternatives they considered were ground up corncobs, peanut shells, popcorn, and things of that nature. Id. In considering these various alternatives, Orton did not perform any research or conduct any inquiry as to the possible health hazards that may have been associated with these materials. Id. Further, at no time in its history did Orton perform any testing on the vermiculite packaging material it used (Exhibit 2, Childress deposition 11/28/07 at 95). At no time did Orton provide any warnings to its customers regarding the hazards of vermiculite contaminated with the Libby amphibole (LA) asbestos. Orton's Corporate Designee, Gary Childress, testified regarding Orton's purchase, use, and sale of packing materials contained in the boxes of Orton pyrometric cones over the course of the company's history. Id at 116. With regard to Orton's purchases of vermiculite, Orton produced as authentic business records a collection of cancelled checks which provided a perspective as to the dates that Orton obtained vermiculite from each supplier. See Exhibit 2, Childress deposition 11/28/07 at 46-67, Exhibit 4, cancelled checks (originally attached to Childress 11/28/07 as deposition exhibit 3), and Exhibit 5, Declaration of Gary Childress, 11/1/07 (originally attached to Childress 11/28/07 as deposition exhibit 2).

W.R Grace / Zonolite	March 1963 – June 1975
J.P. Austin	September 1975 – June 1979
W.R. Grace / Zonolite	September 1979 – early 1982
J.P. Austin	July 1982 – March 1983

In 1983, Orton ceased using vermiculite as its packaging material not for safety reasons, but because the vermiculite "created a lot of dust and [this dust] made the plant dirty." (Exhibit 2, Childress deposition 11/28/07 at 96). At that time, Orton began packaging its cones in styrofoam, a practice which continues to this day (Exhibit 5, Childress declaration).

After purchasing, using, and selling vermiculite for eighteen years, on September 2, 1981, Dale Fronk, Ceramic Engineer for Orton, sent the following letter to W.R. Grace & Co., which reads:

Dear Sirs:

We are required by Federal law to obtain Material Safety Data Sheets for all material we use in the manufacturing of our product, pyrometric cones.

We are currently using and purchasing from your organization Industrial #2 expanded vermiculite. Although this material does not go directly into our product, it is used as a packaging material.

We would appreciate obtaining the above mentioned Material Safety Data Sheets from you along with any other data on Industrial #2 expanded vermiculite which may be valuable to us.

(Exhibit 6, Orton Letter to W.R. Grace & Co., 9.2.81 (originally attached to Childress 11/28/07 as deposition exhibit 5).

W.R. Grace & Co. responded to Mr. Fronk's request on September 25, 1981, which reads:

Dear Mr. Fronk:

This is in response to your recent request for a Material Safety Data Sheet for Industrial Vermiculite #2 which you have been using as packing material.

I am attaching a copy of our M.S.D.S. #Z-140, Industrial Vermiculite, Libby Source- Size #1, #2 & #3.

#3 Expanded Vermiculite is also marketed to consumers as Attic Insulation. When processed for this end-use, it is more intensively screened to remove fines (vermiculite dust and tremolite) and in some cases, it is treated with a binder of the user to airborne tremolite fiber. You may wish to purchase this to limit dust exposure. It is readily available at most building supply houses.

We have prepared the attached Material Safety Data Sheet in a form that is most meaningful to technically aware industrial end-users who will be aware of the responsibilities placed on them by OSHA Regulations.

It has been our experience, handling significant tonnages of expanded products in our own manufacturing plants, that good industrial hygiene practices will limit the airborne exposures of employees to extremely low levels.

(Exhibit 7, W.R. Grace & Co. letter to Orton, 9.25.81 (originally attached to Childress 11/28/07 as deposition exhibit 6).

As referenced in this September 25, 1981 letter, W.R. Grace attached its MSDS for Industrial Vermiculite #2, dated June 10, 1977. This MSDS states:

- Synonyms for this materials include Expanded Libby, Montana Vermiculite;
- Industrial Vermiculite #2 (which Orton used as a packing material) contains naturally occurring contaminant tremolite;
- OSHA defines tremolite as asbestos;
- The physical handling given to expanded Vermiculite can release both airborne fibers and nuisance dust;
- When using Industrial Vermiculite #2, the product should be dampened slightly or one should employ other techniques which control airborne fibers and dust;
- Personal protective equipment should be used to meet exposure limits.

Id.

7. Libby Montana Vermiculite

A vermiculite mine located seven miles northeast of Libby, MT (population ~2,700, with nearly 12,000 in the surrounding area) supplied up to 80% of the world's supply of vermiculite from the early 1900s through 1990 (USEPA, 2012a). Vermiculite expands or pops when heated, creating pockets of air that made the material suitable for use in building insulation and as a soil conditioner. Vermiculite from the Libby mine is contaminated with a toxic form of naturally-occurring fibrous and non-asbestiform amphibole in veins throughout the deposit (Pardee and Larsen, 1929). Approximately 30-40% of the amphiboles are asbestiform and include winchite, richterite, tremolite and magnesioriebeckite; differing in their relative proportions of cations (Mg, Ca, Fe, Na, K) (Meeker et al., 2003; Bandli et al., 2003; Gunter et al, 2003; Sanchez et a., 2008; Gunter and Sanchez, 2009). This amphibole mineral mixture is commonly referred to as Libby amphibole (LA).

Expanded vermiculite was used in numerous commercial products throughout the 20th century. The largest size or grade 1 was used as a loose-fill insulation (attic and wall cavities) in dwellings and buildings and in refrigerators and furnaces (Keigel, 1940). In addition, expanded vermiculite has been used in potting soils and other horticultural products, and it has also been used as a packaging material and was sold by gardening stores as a gardening product (Potter, 1997, USEPA, 2000).

Other uses include an aggregate for insulation plasters, manufacture of fire-resistant, insulating wall-boards and acoustic tile. Very fine sizes were reported to be used as extenders in gold and bronze

paints and inks due to their silver to gold colors that were produced upon expansion (Keigel, 1940). Commercial names for expanded vermiculite include Unifil, Porosil, Zonolite®, and Monokote® (Keigel, 1940; Bandli and Gunter, 2006).

8. Amphibole Occurrence in Vermiculite

An EPA sponsored study (Atkinson et al. 1982) was conducted to determine the amount of asbestiform minerals in vermiculite from three U.S. mining operations; Zonolite Mountain and two South Carolina mines. The estimated content of LA (referred to as tremolite/actinolite) in Libby raw ore at the head feed was reported as 21-26% (by weight) and in processed ore (beneficiated and sized) was reported as 0.3 to 7 % (by weight) (Atkinson et al. 1982). Amphibole asbestos, reported as temolite/actinolite and anthophyllite, was also detected in South Carolina processed and unprocessed vermiculite, but in "substantially lower" concentrations (<1% by weight) for the fibrous phases (Atkinson et al. 1982). Amandus et al. (1987) reported LA concentrations from internal company (W.R. Grace Co.) sampling in raw ore at the head feed and concentrate as 3.5 – 6.4 % and 0.4 – 1.0 % (by weight), respectively.

The amphibole content has also been studied in expanded vermiculite. Moatamed et al. (1986) reported amphibole asbestos concentrations of 0.8 – 2 % and 0.6% for two samples of grade 2 and 3 unexpanded and expanded Libby vermiculite, respectively. A study conducted by the USEPA (2000) revealed asbestos concentrations of 0 to 2.8 % (by weight) in Zonolite brand expanded vermiculite sold in Seattle area retail stores for lawn and garden care use.

Bulk vermiculite samples collected from attic spaces as a component of attic insulation disturbance based studies have revealed amphibole asbestos concentrations from non-detect to 10% (Cowan, 1997; USEPA, 2003a; Ewing et al., 2010: Spear et al., 2012). Forty Montana homes (outside of Lincoln County, MT) containing VAI all revealed the presence of LA asbestos in bulk samples (Spear et al., 2012).

9. Toxicity of Libby Amphibole Asbestos

Occupational exposure to LA is associated with significant increases in asbestosis, lung cancer, and pleural cancer compared to the rest of the U.S. population (Sullivan, 2007). In 2009, the U.S. Environmental Protection Agency (EPA) designated the town of Libby, Montana, a public health emergency (USEPA, 2009). This was the first and only time the EPA has made this determination under the Comprehensive Environmental Response, Compensation and Liability Act (CERCLA).

One of the earliest publications associating Libby vermiculite with pulmonary changes focused on a worker population from a Marysville, Ohio fertilizer plant that had utilized vermiculite from the Libby mine and South Africa (Lockey et al., 1983). This cohort became the basis for the RfC published by EPA (2014a). Significant correlations were observed with respiratory symptoms (shortness of breath and pleuritic chest pain) and cumulative fiber exposures (Lockey et al., 1984). Studies focusing on Libby workers soon followed.

McDonald et al. (1986) included a cohort of 406 men employed at the mine for at least one year prior to 1963 and followed them until 1983. Compared with white men in the U.S., the cohort experienced excess mortality, with standard mortality ratios (SMRs) of 2.45, 2.55, 2.14 for respiratory cancer, non-malignant respiratory disease (NMRD), and accidents, respectively. Standard mortality ratios are

defined as the observed number of cases over expected. The proportional mortality for the four identified mesothelioma deaths was 2.4%. Data collection for a parallel study sponsored by the National Institute for Occupational Safety and Health (NIOSH) was initiated at approximately the same time (Amandus et al. 1987; Amandus and Wheeler, 1987) and included 575 men employed at the mine for a minimum of one year prior to 1970. Similar to the McDonald et al. (1986) study, SMRs were 2.23, 2.43, and 1.44 for respiratory cancer, non-malignant respiratory disease and accidents, respectively (Amandus and Wheeler, 1987). These early occupational-based studies demonstrated strong exposure years/response relationships (McDonald et al., 1986; Amandus and Wheeler, 1987; Antao et al., 2012).

McDonald published additional work in 2004 in which he updated epidemiology data for his original 406 man cohort, following them until 1999 (McDonald et al., 2004). The SMRs reported in this update for lung cancer and non-malignant respiratory disease were 2.40 and 3.09, respectively. The proportional mortality for the 12 identified mesothelioma deaths was 4.21%. An all-cause linear model implied a 14% increase in mortality for mine workers exposed occupationally to 100 f/mL/yr and approximately 3.2% increase for the general population exposed to 0.1f/mL for 50 years. This study also reported overall proportional mortality from mesothelioma in a cohort of vermiculite miners exposed to fibrous amphibole in Libby, Montana, similar to that for crocidolite miners in South Africa and in Australia, and over 10 times higher than that in Quebec chrysotile miners.

An additional NIOSH sponsored study included a cohort of 1,672 Libby miners, millers, and processors in 1982 and followed subjects through 2001 (Sullivan, 2007). Compared with U.S. white men, SMRs for asbestosis, lung cancer, and cancer of the pleura were 165.8, l.7, and 23.3, respectively, with observed dose related increases in asbestosis and lung cancer. An update of the Sullivan (2007) cohort was published recently (Moolgavkar et al., 2010), revealing similar SMRs to Sullivan. In addition, estimates of relative risk for lung cancer, non-malignant respiratory disease, and total mortality were 1.2, 1.4, and 1.06, respectively, with 95% confidence intervals of [(1.06, 1.17), (1.09, 1.18), and 1.04, 1.08)] (Moolgavkar et al., 2010).

One of the latest updates regarding vermiculite worker mortality (Larson et al., 2010), with a cohort of 1862 Libby miners, demonstrated a clear exposure response relationship between cumulative Libby amphibole fiber exposure and asbestosis, lung cancer, mesothelioma, and NMRD mortality. A limitation noted for earlier epidemiology studies evaluating lung cancer SMRs in Libby mine and mill workers was the lack of control for cigarette smoking. Bias analysis revealed that cigarette smoking had minimal impact on the exposure response relationships reported in this study (Larson et al., 2010; reviewed by Antao et al., 2012). An additional conclusion from this study was the association between Libby amphibole fiber exposure and cardiovascular mortality based on a rate ratio of 1.5 with a 95% confidence interval of 1.1 to 2.0 (Larson et al., 2010).

A follow-up to the Lockey et al. (1984) Marysville, Ohio fertilizer plant study revealed pleural changes in 28.7% of the cohort (Rohs, et al., 2008). As noted previously, this cohort was the basis for the proposed Libby amphibole RfC. Pleural changes were originally reported in 2.2% of the overall cohort and 8.4% of the highest cumulative fiber exposure group (Lockey et al., 1984). The study is significant in that the

cohort was based on exfoliation plant workers outside of Libby, MT, with relatively low cumulative fiber exposure levels compared to those described in the Libby mine and mill worker studies.

In addition to epidemiology studies that considered Libby mine and mill workers, research has also included studies evaluating ARD mortality among Libby community members. A cross-section interview and medical testing of 7,307 persons who had lived, worked or played in Libby for at least six months prior to 1991 was conducted in 2000 and 2001 by the Agency for Toxic Substance and Disease Registry (ATSDR) investigators (Peipins et al., 2003). Of the 6,668 participants > 18 years of age who received chest radiographs, pleural abnormalities and interstitial abnormalities were observed in 17.8% and < 1% of the participants, respectively. Participant interviews revealed that the factors most strongly associated with pleural abnormalities were being a former vermiculite mine or mill worker, age, having been a household contact of a former vermiculite mine or mill worker, and being male (Peipins et al., 2003).

In 2008, a clinical and exposure summary report for 11 individuals diagnosed with mesothelioma who were not Libby mine or mill employees was published (Whitehouse et al., 2008). All cases were non-occupationally exposed individuals. The authors reported pleural and peritoneal mesothelioma associated with secondary exposure to LA and concluded that exposure most likely resulted from Libby amphibole contamination in the community, the surrounding forested area, and areas in proximity to the Kootenai river and railroad tracks that were used to transport vermiculite concentrate (Whitehouse et al., 2008). The mean LA occupationally related mesothelioma latency period has been reported as 35 years (Case, 2006). The latency period reported for these non-occupational cases was 13-67 years from the first known exposure (Whitehouse et al., 2008).

In terms of both occupational and non-occupational mesothelioma cases, current mortality figures indicate one new case per year in Lincoln, County, Montana (McDonald et al., 2004; Case, 2006; Whitehouse et al., 2008). Lincoln County has the third highest age-adjusted mesothelioma death rate in the nation with a rate of 56.1 per million population (NIOSH, 2008).

A community study of Libby residents who were children (< 18 years) when the vermiculite mine closed in 1990 revealed a positive association between self-reported respiratory outcomes and certain activities with potential Libby amphibole exposure pathways (Vinikoor et al., 2012). Of the 1,003 study participants, 10.8% reported usually having a cough, 14.5% reported experiencing shortness of breath when walking up a slight hill or hurrying while on level ground, and 5.9% reported having coughed up bloody phlegm in the past year. Handling vermiculite insulation was positively associated with three of the four outcomes examined compared with never handling vermiculite insulation. No association was found between vermiculite insulation in the home and respiratory symptoms and no association was found between any of the activities and abnormal spirometry (Vinikoor et al., 2012).

A community study was conducted in a densely populated urban residential neighborhood in Minneapolis, Minnesota where an expansion facility processed Libby vermiculite ore from 1938 to 1989 (Alexander et al., 2012). In addition to commercial vermiculite products such as Zonolite® insulation and Monokote® fireproofing, the facility produced a waste material reported by the Minnesota Department

of Health to contain 10% amphibole asbestos (Alexander et al., 2012). The waste product was piled on the property and offered to the community for use in gardening, driveway fill materials, etc. The prevalence of pleural abnormalities obtained for the 461 participants was 10.8%. The odds ratio associated with direct contact with vermiculite ore waste or ever playing in waste piles and pleural abnormalities was 2.78 (95% CI: 1.26, 6,10) and 2.17 (95% CI: 0.99, 4.78) when adjusted for background exposure. Although this study was conducted outside of Libby, MT, the results suggest that community exposure to Libby vermiculite is associated with measurable effects (Alexander et al., 2012).

In addition to pulmonary based ARD, rates of systemic autoimmune diseases (SAID) have been evaluated in the Libby community. A follow-up case-control study was conducted among the participants in the 2000/2001 ATSDR study (Peipins et al, 2003) with cases including subjects that reported one of three (SAIDs) in the initial screening; systemic lupus erythematosus, scleroderma, or rheumatoid arthritis, and controls including subjects in the initial screening that responded negatively to questions regarding SAIDs (Noonan et al., 2006). Odds ratios among former Libby mine and mill workers > 65 years of age of 2.14 (95% CI, 0.9-5.1) for all SAIDs and 3.23 (95% CI, 1.31 7.96) for rheumatoid arthritis, suggest that LA exposure is associated with SAIDs (Noonan et al., 2005). Increasing SAIDs risk estimates were reported for participants with relative increases in reported vermiculite exposure pathways.

These epidemiologic studies demonstrate clear and significant increases in asbestos-related disease (ARD), including asbestosis, lung cancer, and mesothelioma among former mill and mine workers. In addition, ARD has been observed in area residents with no direct occupational exposures. The most common health outcome among Libby residents and others with low lifetime cumulative fiber exposure levels are pleural changes.

10. Exposure Pathway

An exposure pathway is the process by which an individual is exposed to contaminants originating from a contamination source. An exposure pathway consists of the following five elements: (1) a *source* of contamination; (2) a *media* such as air or soil through which the contaminant is transported; (3) a *point of exposure* where people can contact the contaminant; (4) a *route of exposure* by which the contaminant enters or contacts the body; and (5) a *receptor population*. A pathway is considered complete if all five elements are present and connected. It is currently believed that in Libby the most important of these exposure pathways is the inhalation of air in the immediate vicinity of an active soil disturbance that causes a release of LA fibers from soil into the air (ATSDR, 2003a).

Grace Webster had a complete pathway of exposure to Libby Amphibole Asbestos (LA) through his work involving Orton Company cones packaged in vermiculite. The source of contamination was vermiculite. The disturbance of vermiculite contaminated with LA through her work activities dispersed asbestos fibers into the air and into her breathing zone. Grace Webster performed ceramics work using Orton cones for over 30 years and all of her students performed the same work with the kilns and pyrometric cones. On an average week, Grace Webster used dozens of pyrometric cones in her ceramics classes. She was exposed to visible dust from the vermiculite when she opened the cardboard boxes and sifted

through the vermiculite to get the pyrometric cones. The dust from the vermiculite packing would also be carried out on the cones from the boxes and this dispersed visible dust into her breathing zone. She was also exposed to visible dust from the vermiculite when she would dump the boxes of cones onto a tray to access the cones. Grace Webster used brooms to sweep up the dust and debris from the vermiculite packing material and this dispersed visible dust into her breathing zone. Throughout her career, Grace Webster used kilns and pyrometric cones packaged in vermiculite, and she continued to be exposed to visible dust containing LA from the same vermiculite packing material on a regular basis.

Asbestos residence time in the air is determined primarily by particulate thickness; however it is influenced by other factors such as length and static charge. The average thickness of LA fibers is 0.4 µm and ranges from approximately 0.1 to 1.0 µm. The suspension of LA in air is measured in "half times" which is the amount of time it will take 50% of LA particles to settle out of the air column. A particle with a thickness of 0.5 µm has a half time of approximately two hours, assuming the source of disturbance has been removed (CDM, 2009).

Larger particles will settle faster; a particle of 1 µm has a half time of about 30 minutes. Smaller LA particles may stay suspended for significantly longer. The typical half time for a 0.15 particle is close to 40 hours (CDM, 2009).

Activity-specific testing found that the half-time of LA suspended by dropping vermiculite on the ground was about 30 minutes. LA suspended from disturbing vermiculite insulation settled within approximately 24 hours (CDM, 2009).Once suspended, LA moves by dispersion through air. LA concentration will be highest near the source and will decrease with increasing distance.

11. Fiber Re-entrainment

Because of their shape and small size, asbestos fibers, particularly those of respirable dimensions, remain airborne for hours once they are introduced into the air. Once they are airborne the asbestos fibers will drift long distances from their source. Movement and air turbulence causes fibers that have settled out of the air to be reintroduced (re-entrained) into the air and to drift long distances from their source. In addition, the human traffic on a worksite can also be expected to disburse asbestos throughout the entire work area. For this reason, asbestos fibers do not respect work areas or job classifications. It has been repeatedly demonstrated that a source of asbestos emission in the air puts everyone in the general vicinity (bystander exposure) at risk. Because of the microscopic size of asbestos fibers, and their aerodynamic properties, typical housekeeping activities such as sweeping tend not to remove that asbestos from the plant. Rather, such activities have the effect of stirring up and re-entraining the asbestos that is in the location, ensuring that it is available for inhalation by workers in the vicinity.

12. Evidence of Fiber Release From Source Media Containing < 1% Asbestos

In summary, research by the U.S. EPA and others has shown that disturbance of matrices (e.g., soil, vermiculite insulation) containing asbestos concentrations identified by the lower detection limits of PLM—well below 1% asbestos by weight, can generate potentially hazardous exposures.
Fiber release studies in chambers or activity based sampling performed by workers equipped in personal protective equipment mimic the various types of activities conducted by people to determine whether

asbestos fibers in a source media could be released into the breathing zone of individuals conducting these activities. The concentrations of asbestos fibers measured by personal and area monitoring during the various planned activities are used to estimate risks associated with these activities.

EPA 2004. Memorandum Clarifying Cleanup Goals and Identification of New Assessment Tools for Evaluating Asbestos at Superfund Cleanups. Office of Solid Waste and Emergency Response. OSWer 9345.4-05.

> Memorandum stated that regions should not assume that materials containing less that 1 percent asbestos do not pose an unreasonable risk to human health. The 1 percent threshold concept was related to the limit of detection for the analytical methods and the 1 percent threshold in soil/debris as an action level may not be protective of human health. The 1 percent threshold is not risk-based and an accurate exposure value could only be determined through site sampling techniques that generate fibers from soil and bulk samples. Recent data from the Libby site and other sites provide evidence that soil/debris containing significantly less than 1 percent asbestos can release unacceptable air concentrations of all types of asbestos fibers (i.e., serpentine/chrysotile and amphibole/tremolite). The most critical determining factors in the level of airborne concentrations are the degree of disturbance and the presence of complete exposure pathways.

Libby Asbestos Superfund Site Residential and Commercial Properties Operable Unit 4 Libby, Montana Remedial Investigation Report Contract No. W9128F-11-D-0023 Task Order No.: 0003 and 0007 June 2014

> *LA Levels in Soil that is Non-detect by PLM*
> The EPA uses PLM-VE to estimate levels of LA in soil in Libby. This is a semi-quantitative method that reports a sample as non-detect when the microscopist cannot observe any LA in the sample. However, from the studies of outdoor soil disturbance, it is evident that soils that are non-detect can release LA fibers to air. For this reason, the EPA used more powerful electron microscopy methods to estimate the average level of LA in soils that were reported as non-detect by PLM-VE. The results were variable between samples, but the average LA concentration was approximately 0.05% by mass.

Januch J, McDermott K (2004). Study of Asbestos Contamination of Former Vermiculite Northwest/W.R. Grace Vermiculite Exfoliation Facility. US EPA Region 10, Office of Environmental Assessment, Investigation and Engineering Unit, Seattle, Washington.

> EPA Region 10 conducted a three phase study at the Spokane vermiculite exfoliation plant to determine if asbestos fibers in the soil at the site could become airborne when the soil was disturbed. Twelve soil specimens were collected from the site and eleven were agitated inside a laboratory enclosure equipped with air monitoring equipment. Ten of the eleven soil specimens contained asbestos that became airborne when the soil was agitated. Filters used for collection of air samples were analyzed with a transmission electron microscope (TEM) and were found to contain asbestos, with concentrations of asbestos in the air ranging from 0.051 fibers per cubic centimeter (f/cc) to 10.713 f/cc. Air samples were collected while performing property maintenance and excavation tasks at two locations on-site. Samples analyzed using TEM showed

concentrations of asbestos ranging from 0.010 f/cc to 0.045 f/cc of air. Several asbestos fibers were also detected in filters from stationary air monitors.

EPA (December 2001), Weis Memo to Paul Peronard: Re: "Amphibole mineral fibers in source material in residential and commercial." Libby Site, Montana. USEPA Region 8; December 20, 2001.

Reported release of asbestos fibers, from soil containing <1% to 6% asbestos, during removal activities by workers at the Screening Plant. It was demonstrated that concentrations significantly above the OSHA occupational limit of 0.1 f/cc were detected by personal air monitors in the breathing zone of workers, during routine activities including soil bagging and sweeping floors for most size classes as measured by transmission electron microscopy analysis. It is important to recognize that occupational exposure standards for asbestos are not generally applicable or protective for residents or workers in non-asbestos environments because occupational standards are intended to protect individuals who **a)** are fully aware of the hazards of the occupational environment, **b)** have specific training and access to protective equipment such as respirators and/or protective clothing and, **c)** actively participate in medical monitoring (USEPA 1995). None of these conditions apply to residents or to workers at typical commercial establishments. Thus, simple compliance with the OSHA standards is not evidence that exposure levels are acceptable in a home or in a non-asbestos workplace. Indeed, risks to residents or workers occur at exposure levels substantially below the OSHA workplace standards. It should be noted that according to OSHA estimates, 0.1 f/cc limit is recognized as being associated with significant risk (of 3.4 additional cancers per 1000 individuals) to workers and risks to residents could be higher. For example, the concentrations were:
< 0.61 f/cc for fibers of length = 0.5 to 5 um; diameter <0.5 um
3.055 f/cc for fibers of length = 5-10 um; diameter <0.5 um
1.222 f/cc for fibers of length >10 um; diameter <0.5 um
1.222 f/cc for fibers of diameter >0.5 um

Colorado Department of Public Health and Environmental, Asbestos-contaminated Soil Guidance Document. Hazardous Materials and Waste Management Division. Revised April 2007.

Several studies using a variety of approaches, including the state of the science, for the release of asbestos fibers from significantly <1% asbestos in soil/debris demonstrated that all types of asbestos fibers can be released into the air or breathing zone during soil disturbing activities resulting in unacceptable risk that is significantly above acceptable cancer risk level of 1 in a million at 0.000004 ($4X10^{-6}$) f/cc (EPA IRIS), and even above the OSHA limit of 0.1 f/cc, in some cases.

EPA 2000. Office of Prevention, Pesticides and Toxic Substances (7401). EPA 744-R-00-010. August 2000.

To simulate indoor use of vermiculite garden products and measure indoor air asbestos concentrations, a 10'x10'x10' containment was constructed to represent a homeowner's garage or small greenhouse. None of the products tested using PLM had detectable levels of asbestos. Seventeen of the 36 products had detectable asbestos using TEM analysis, with five products

containing greater than 0.1 percent asbestos by weight and ranging from 0.13 to 0.7 percent actinolite asbestos. TEM analysis during the use of Zonolite® Chemical Packaging Vermiculite generated airborne asbestos concentrations ranging from non-detect to 0.0769 s/cc actinolite >5 um in length in indoor area monitors, and 0.4171 to 0.6594 s/cc actinolite >5 um in length in the personal samples.

Versar, Inc. Pilot study to estimate asbestos exposure from vermiculite attic insulation. Final draft 21 May 2003. Washington DC: US Environmental Protection Agency; 2003.

Under contract to the US EPA Versar, Inc. has also conducted a series of studies to characterize asbestos exposures from vermiculite attic insulation. They found significantly increased airborne concentrations when the vermiculite attic insulation was directly disturbed.

Ewing WM, Hays SM, Hatfield R, Longo WE, Millette JR. Zonolite Attic Insulation Exposure Studies. Int J Occup Environ Health. 2010;16:279-90.

Demonstrated that many routine cleaning, maintenance, and remodeling activities that disturb Zonolite attic insulation can generate significant airborne amphibole asbestos exposures and the OSHA excursion limit for asbestos of 1 f/cc during any 30-minute period was often exceeded. Depending on the length of the work, the OSHA eight-hour permissible exposure limit (PEL) would often have been exceeded. The authors stated: “When such work in attics are performed by homeowners, the OSHA regulations do not apply.”

Addison J., Davies L.S.T., Dwaneson A., Willey R.J. The release of dispersed asbestos fibres from soils. Historical Research Report. Research Report TM/88/14 (1988).

Study demonstrated release of asbestos fibers (>OSHA occupational limit of 0.1 f/mL) from soils containing 0.001% asbestos. Chrysotile 0.001% in intermediate soil = 0.23 f/mL by scanning electron microscopy; Chrysotile 0.1 % in clay = 1.17 f/mL by scanning electron microscopy; Chrysotile 1% in intermediate soil = 48.5 f/mL by scanning electron microscopy.

Addison et al. (1988) recommended “that soils containing more than 0.001% asbestos are regarded as being capable of generating airborne fibre concentrations in excess of 0.1 f ml-1 (the OSHA workplace standard) and that precautions to protect the workforce by wetting the soil, providing respiratory protection etc., are taken”.

Addison, J. Vermiculite: a review of the mineralogy and health effects of vermiculite exploitation. Reg. Tox. Pharm. 21: 397-405 (1995).

It would be necessary therefore to take action specifically to control for the asbestos emissions if soils containing higher levels than 0.001% asbestos were to be handled without significant health risks. Asbestos, if present in vermiculite, is likely to behave in a similar fashion; with the asbestos loosely dispersed and readily available for release into the air. Even relatively gentle handling of the vermiculite would abrade the friable asbestos, splitting fiber bundles, and

adding to the released fibers. Thus, even though the carcinogens legislation may impose only a 0.1% limit for packaging and labeling, the vermiculite industries would be advised to establish their own target limit of 0.001% for amphibole asbestos. Most current supplies of vermiculite could still meet this standard (Addison, 1995).

Gordon RE, Fitzgerald S, Millette J. Asbestos in commercial cosmetic talcum powder as a cause of mesothelioma in women. Int J Occup Environ Health. 2014 Oct;20(4):318-32).

Three laboratories confirmed in multiple tests the presence of asbestiform anthophyllite and asbestiform tremolite in talcum powder products. All 50 samples of the product analyzed by Laboratory A showed that all the samples contained asbestos fibers; 80% containing only anthophyllite asbestos, 8% only tremolite asbestos, 8% anthophyllite and tremolite asbestos, and 4% anthophyllite, tremolite, and chrysotile asbestos.

PLM analysis performed by Laboratory C showed that the samples contained both platy and fibrous talc, less than 1% by volume of the PLM. One of the talcum powder samples was found to contain 4% anthophyllite by XRD. By TEM analysis all nine samples were positive for amphibole asbestos, primarily anthophyllite, and were confirmed with zone-axis electron diffraction measurements. At least five asbestos fibers per sample were recorded in each sample, with concentrations ranging from 0.004 to 0.9% by weight and from 3 to 200 million asbestos fibers per gram of fibers greater than 0.5 um in length with at least a 5:1 aspect ratio.

Glove box simulation testing was performed by Laboratory B and full chamber testing was performed by Laboratory C for releasability of asbestos into the air. The asbestos concentration in the breathing zone of the talc user during shaker application was 1.9 F/cc. The asbestos concentration in the breathing zone of the bystander was 0.5 F/cc. The asbestos concentrations in the breathing zone of the talcum powder user during puff application were 5 and 3.5 F/cc. The short term sample in the breathing zone of the applier had an asbestos concentration of 13 F/cc. The asbestos concentrations in the breathing zone of the bystander were 4.9 and 3.5 F/cc.

Full chamber testing in a bath-sized room performed by Laboratory C confirmed the finding for asbestos fiber release during the glove box testing, showing significant concentrations of anthophyllite, tremolite and occasionally chrysotile.

MAS Project 14-1852. Below the Waist Application of Johnson & Johnson Baby Powder. William Longo et al. September, 2017.

Approximately 4 grams of baby powder were applied to the lower body of an investigator to determine the potential exposure levels of an individual to amphibole asbestos fibers while applying Johnson & Johnson baby powder. Air samples were analyzed by NIOSH 7400 PCM and NIOSH 7402 TEM.

During the Johnson's Baby Powder application procedure, personal breathing zone and area samples were collected over a period of 5 minutes. Samples analyzed by NIOSH 7400 PCM analysis found that four personal sample results ranged from 3.85 f/cc to 5.86 f/cc with an average mean of 4.52 f/cc. Area sample results were 0.28 f/cc to 0.58 f/cc with an average mean of 0.41 f/cc.

Four personal PCM filters analyzed by NIOSH 7402 TEM method showed the percent tremolite asbestos fiber concentration ranging from 42.9% to 76.9%, resulting in a PCM equivalent range of 1.81 f/cc to 4.51 f/cc with an average mean of 2.57 f/cc.

NIOSH 7402 TEM method was also used to quantify the airborne fibrous talc concentrations in four personal air samples, and the fibrous talc concentrations ranged from 0.60 f/cc to 1.86 f/cc with an average mean of 1.51 f/cc.

MVA 17 July 2018. Report of Results: MVA12466. Analysis of Vermiculite Packing Material of Orton Pyrometric Cones. Sean Fitzgerald. Scientific Analytical Institute. March 14, 2019.

Ten of the samples containing vermiculite packing materials were found by TEM to be positive for Libby amphiboles. Nine of the fourteen vermiculite packed Orton Pyrometric Cones boxes contained fibers consistent with Libby amphibole mineral fibers. PLM results of six samples confirmed the presence of tremolite/actinolite asbestos.

Asbestos releasability was assessed by air sample analysis during simulation of product use, consistent with normal product use, in a controlled environment. Amphibole fibers and bundles were found with characteristic asbestiform morphology, crystalline structure by SAED, appropriate ratio of elements to chemical formulas of those minerals as determined by EDS. Average concentrations of airborne asbestos fiber structures per cubic centimeter (str/cc) of the product ranged from 0.416 to 11.72 Structures/cc.

13. Knowledge of Asbestos Hazards in Scientific and Occupational Medicine Literature

The asbestos industry started in the 1870's with asbestos components used in insulation, materials to withstand high temperatures, and numerous other industrial applications (Castleman, 1996). The earliest case study reports potentially linking asbestos exposure with pulmonary disease were published in Great Britain. The Lady Inspectors of Great Britain provided some of the earliest documentation of asbestos related health hazards when they included asbestos work as one ofthe four dusty occupations under investigation in 1898 due to "injury to bronchial tubes and lungs medically attributed to employment" (Deane, 1898 as reported by Castleman, 1996 and Greenburg, 1999). In the early 1900's, there were case study reports of pulmonary disease in asbestos plant workers in Great Britain, France, Italy, and Germany (Castleman, 1996).

In the U.S. in 1917, Pancoast, Miller and Landis, in an attempt to characterize increased thickening observed in individuals examined for tuberculosis and other conditions (pneumoconiosis), examined the chests of workers engaged in dusty occupations in a roentgenologic study. Fifteen asbestos workers were included in the 137 individuals examined. Fibrous tissue and localized fibrosis was observed in asbestos workers (Pancoast, et al., 1918).

By 1918, Hoffman, a statistician with the Prudential Life Insurance Company, recognized asbestos work as a hazardous trade. "In the practice of American and Canadian life insurance companies, asbestos workers are generally declined on account of the assumed health-injurious conditions of the industry" (Hoffman, 1918).

While there was documentation of pulmonary disorders associated with asbestos exposure in the early 20th century, Dr. W.E. Cooke, an English pathologist, was the first to describe fibrosis of the

lungs due to asbestos exposure in medical literature (Cooke, 1924 and Cooke, 1927). The subject of Cooke's papers was a 33 year old female that worked in the spinning room of a Rochdale asbestos company. Additional case reports soon followed, including work by Thomas Oliver, an M.D. who focused on occupational disease. His work, which was published in British and U.S. medical journals (Oliver, 1927a; 1927b) described asbestos manufacturing as a "familial" occupation with generations of females. Two of these women who worked in the asbestos industry were the subject of his case studies describing pulmonary asbestosis (Oliver, 1927a; 1927b). While Cooke is cited in some textbooks as being the first to coin the term "asbestosis", some credit it to Oliver (Bartrip, 2003). Gloyne (1933) described the microscopic appearance of asbestosis and anatomical changes observed with various stages of the disease.

Soon after the Cooke and Oliver publications, Dr. Merewether, as British medical inspector, was instructed to determine whether or not a health risk was truly present in the asbestos industry. Meriwether and Price examined 363 asbestos workers who did not have previous work in dusty occupations. Variables considered included length of employment and dustiness of the job. Twenty six percent of those examined had asbestosis. When subjects working less than five years were excluded, the incidence of asbestosis increased to 35 percent. The number of years employed in the asbestos industry was a primary risk factor for disease (Merewether, 1930a; Merewether, 1930b). In addition, to illustrating the incidence of asbestosis in the asbestos industry, Merewether and Price discussed the importance of dust suppression tactics, focused on the importance of clinical exams in diagnosis of disease, and associated the inhalation of asbestos dust with a fatal disease. In addition to British publications, Merewether's work was published in the U.S. Journal of Industrial Hygiene (Merewether, 1930b).

Although the "deadly nature of asbestos dust was widely known in medical, public health and industry circles in the 1930s, the consumption of asbestos rebounded strongly after the worst years of the great depression" (Castleman, 1996).

Drinker and Hatch (1936) described the disabling pneumoconiosis associated with asbestos exposure as asbestosis with characteristic symptoms and chest X-rays in their book "Industrial Dust." The same year, the Illinois Workers Compensation Act recognized asbestosis as a compensable occupational disease and required employers in the state of Illinois including John Crane, Inc. and Crane Co., to affirmatively elect whether to pay compensation under the Act or continue to be liable in the tort system.

In 1938, an epidemiologic study of 541 workers in four asbestos textile plants in North Carolina was published (Dreessen et al., 1938). Along with job classifications and work history data, dust sampling was conducted in the plants. The primary variables identified in occupational groups affected by asbestosis were average concentration of dust exposure and length of employment. Since only three cases of asbestosis were observed at dust exposure concentrations less than 5 million particles per cubic feet (5 mppcf), Dreessen et al., (1938) proposed that "if asbestos dust concentrations were kept below this limit new asbestosis cases would not appear." This was the first federally proposed recommended exposure limit for asbestos. The Dreessen et al., (1938) study also described the discrepancies between U.S. and British factories in terms of dust control practices, with British factories employing greater dust control measures. Also in 1938 the

Pennsylvania Workers Compensation Act recognized asbestosis as a compensable occupational disease.

In the 1930s through the early 1940s, individual case reports describing lung cancer in the presence of pulmonary asbestosis were frequently published in literature (Lynch and Smith, 1935; Gloyne, 1935; Gloyne 1935b; Egbert and Geiger, 1936; Gloyne, 1936; Lynch and Smith, 1939; Holleb and Angrist, 1942). Subjects in the 1930s case reports worked as weavers, spinners, and misc. areas of asbestos plants. In the 1940s, pipe insulators were included in these case reports (Holleb and Angrist, 1942). In a summary of three additional case reports, Hamburger (1943), noted that the total number of case reports describing the co-incidence of primary carcinoma of the lungs and pulmonary asbestos was up to 19. While Lynch and Smith (1939) suggested that asbestosis was a predisposing factor in carcinoma ofthe lung, Hamburger (1943) concluded that "statistical calculations and morphologic studies did not reliably answer the question of whether asbestosis has to be considered as an etiologic factor in pulmonary carcinoma."

In a 1942 U.S. publication, Hueper (1942) cited case reports to suggest occupational causation of an asbestos cancer hazard. However, Hueper further noted the need for more clinical, pathological, and statistical studies to define cancer risks associated with asbestos exposure. A year later, in Germany, "asbestos in combination with lung cancer" was considered a state compensable occupational disease (Castleman, 1996; Literature of Industrial Hygiene Abstracts, 1944).

In 1944, asbestos was identified as a physical or chemical agent known to or suspected of causing occupational cancer in the Journal of the American Medical Association (JAMA, 1944). Occupational cancers were defined as those "elicited by exposure to the agents in the course of regular occupations." The same year, the Virginia Workers Compensation Act recognized asbestosis as a compensable occupational disease.

In 1949, Wyers, the medical advisor to the South African crocidolite Cape asbestos fields, recommended that, for humanitarian reasons, additional preventive action might be implemented promptly rather than waiting for further evidence of the carcinogenicity of asbestos (Greenberg, 1999; Wyers, 1949). The same year, the Journal of the American Medical Association, published an editorial in its monthly journal which concluded that asbestosis was now a proven cause of lung cancer. (JAMA 8/13/49 “Asbestosis and Cancer of the Lung”, p. 1218)

In 1951, Vorwald et al. published a summary of case studies conducted at the Saranac lab describing experiments conducted on animals exposed to various kinds of asbestos dust. Inhalation and intratracheal injection techniques were used on guinea pigs, rabbits, cats, dogs, rats and mice to investigate tissue reactions. Vorwald et al. concluded that the rabbit, guinea pig and rat animals, but not the mouse and dog, developed peribronchial lung fibrosis similar to human asbestosis after being exposed to chrysotile asbestos. In addition, he concluded that long fibers (20 to 50 microns) were essential in the production of this fibrosis and that as the asbestos concentration increased, the pulmonary reaction time decreased. While chrysotile asbestos was the primary mineral discussed in Vorwald's comment and summary, it is important to note that similar peribronchial lung fibrosis observations were made with amphibole mineral species,

including tremolite (Vorwald, et al., 1951 (Tables 15 and 16)). At the time of Vorwald's publication, tremolite was reported to be the primary amphibole contaminant within the Rainy Creek Complex, the source of Zonolite insulation (Pardee and Larsen, 1929; Bassett, 1959; Boettcher, 1966).

Doll (1955) is credited with the first published epidemiologic study of lung cancer mortality and asbestos workers. Doll noted that from 1935 to the time ofhis publication, among the study group, there were 61 cases of lung cancer in individuals with asbestosis. His review of 105 mortality records for Turner Brothers asbestos workers identified 18 lung cancer cases, 15 in association with asbestosis. He followed up with a group of 113 workers that were employed for a minimum of 20 years in scheduled areas of the factory (those scheduled under 1931 British Asbestos Industry Regulations as being "dusty"). There were 11 lung cancer deaths among this group, (all with asbestosis). Doll also defined an average period between the worker's initial employment in the asbestos industry and death. By 1956, New York (Buffalo Pumps' home state) required employers to prevent "air contamination" from asbestos and other hazardous substances in the workplace.

The text "Asbestos-From Rock to Fabric" from the Textile Institute, 1956, was published to address the need for a comprehensive work on asbestos, including a chapter on asbestos gaskets and packing. This book discussed all of the various uses of asbestos, asbestos dust and asbestos dust control, asbestosis and lung cancer, and referenced recent medical and industrial hygiene literature of interest including the following and many others:

- Vorwald (1951) where the "Inhalation or injection of asbestos fibres caused peribronchiolar fibrosis similar to human asbestosis in guinea-pigs, rats, rabbits, and cats, but not in dogs or mice."
- Carcinoma and asbestosis of the lung in Owen in the British Journal Cancer (5, 382-3) where Asbestosis of the lung was found 20 years after the patient had worked in an asbestos factory for one year (including a photograph of a pleural cancer).
- Asbestosis (Sayers and W. C. Dreesen 1939) in the American Journal of Public Health, stating "Data so far obtained indicate that 5 million particles/cu.ft. is the maximum safe concentration."
- Lung carcinoma caused by asbestos inhalation Boemke (1953) in Med. Monatsschr where "The relation between fibrotic changes caused by asbestos deposits in the lung and carcinoma is discussed."
- The pathology of diseases due to the inhalation of dust (Boemke, 1947) in Med. Monatsschr where "pulmonary disorders due to Aluminium dust, and asbestosis are discussed. Attention is called to the association of asbestosis with pulmonary carcinoma, the latter almost always taking the form of carcinoma of the pavement epithelium."
- Mortality from lung cancer in asbestos workers (Doll, 1955) in Brit.].Ind. Med. where "Among 105 persons who had been employed at one asbestos works, and for whom the cause of death was determined at autopsy, 18 showed lung cancer. Of these 18, 15 also showed asbestosis..."
- Destruction of mineral particles and fibres in the lung after exposure to asbestos dust (Knox and Beattie, 1954) in Arch. Ind. Hyg. Occ.Med. where "Examination of lungs from

27 cases where asbestos exposure showed that the greatest proportion of particles were in the 5 to 25).!. Range" (Carroi-Porczynski, 1956).

Wagner et al., (1960) discussed 33 histologically proven case studies of mesothelioma of the pleura. An association with asbestos originating from the South African Cape crocidolite asbestos field was observed in 28 of the cases, while four cases were associated with the asbestos industry. While this was the first major summary of mesothelial case studies observed, individual cases, (one to two workers) were reported as early as 1933 (Gloyne); however, asbestos was not reported to be associated with pleural mesothelioma until 1943 (Wedler). In terms of the association between asbestosis and mesothelioma, Cartier (1952) described this relationship as "minimal" and "none" in a summary of two Canadian chrysotile case studies of mesothelioma. O'Donnell (1966) studied multiple mesothelioma cases in a plant that "used the chrysotile type of asbestos fiber almost exclusively" and found no relationship between asbestosis and mesothelioma.

In 1963, a study of asbestos insulation workers in New York and New Jersey revealed 10 mesothelioma deaths (Selikoff et al. 1963). While this and subsequent publications (Selikoff et al., 1964; Selikoff and Hammond 1965-66) expanded asbestos exposure and disease research from asbestos mining and asbestos factory workers to those that used asbestos-containing materials in their occupations (Bartrip, 2003), asbestosis in workers who handled asbestos-containing products was observed as early as the 1930's.

Asbestosis in workers who handled asbestos-containing products was observed as early as the 1930's. Merewether and Price (1930) described the risk of asbestos dust exposures for secondary users of asbestos-containing products. Ellman (1934) reported asbestosis in an insulation worker, and more than 20 cases of asbestosis among secondary users product of asbestos products were reported by 1949 including cases among workers applying spray insulation on ships. Frost et a!. (1956) reported high risk of pleural calcification and asbestosis among insulators. In the 1956 annual report of the Chief Inspector of Factories, lagging or insulation work was recognized as hazardous (Selikoff and Hammond, 1965).

Pleural mesothelioma in insulation workers were reported as early as 1947 (Massachusetts General Hospital, 1947; Vander Schoot, 1958; Eisenstadt and Wilson, 1960). McVittie (1965) reported the occurrence of asbestosis among secondary users of asbestos products, and he reported the occurrence of asbestosis among maintenance workers, noting that maintenance work was an important cause of asbestosis. MeVittie referred to the Federal and Workshops Act 190l in which the Secretary of State issued a certificate to the effect that "the manipulation of asbestos and the manufacture and repair of articles composed wholly or partly of asbestos and processes incidental thereto are dangerous." McVittie (1965) also referred to the Industrial Injuries Act of 1930 where compensation for pneumoconiosis was provided for "the working or handling of asbestos or any admixture of asbestos" and "the manufacture or repair of asbestos textiles or other articles containing or composed of asbestos." Lieben (1967) reported mesotheliomas from brief exposures to asbestos, from living in the vicinity of an asbestos plant, and from working with asbestos insulation products.

Selikoff and Hammond (1965) reported that 86% of asbestos insulators with more than 20 years since first employment had radiographic evidence of asbestosis, and insulators were also shown to be at high risk of lung cancer and mesothelioma (Hammond et al., 1965). Craft workers who have been found to be at risk of asbestosis have included insulators, welders, plumbers, pipefitters, bricklayers, carpenters, builders, hod carriers, iron workers, and aluminum and chemical plant workers. One report by Williams (1942) described asbestosis in an aluminum plant worker who wore an asbestos apron and gloves and a more recent paper by Samimi (1981) assessed the magnitude of fiber emission from asbestos gloves. Between 1946 and 1964 there were seven epidemiological reports describing asbestosis and one describing excess lung cancers in workers using asbestos- containing products. Abstracts, review articles, and medical textbooks mentioned the asbestosis hazard to insulators and/or asbestos product users beginning in the 1930's. Several of these reports explicitly included asbestos product users as being at risk to lung disease, as well as asbestos miners and textile plant workers (Hoffman, 1918; International Labour Office, 1930; Soper, 1930; Willson, 1931; Holleb and Angrist, 1942). The hazards of asbestos product use were also reported in engineering and safety publications, trade magazines, and government reports in the early 1900's (See Castleman, 1996).

By 1958, the danger of exposure to asbestos dust from asbestos-containing materials, including valve packing, gaskets, boiler lagging and pipe covering, protective clothing, shielding materials and brake linings, and asbestos cement products, was recognized and included in the American Industrial Hygiene Association's Hygienic Guide series 58.

Mesotheliomas have been documented repeatedly in workers at friction-product factories and among brake mechanics, their wives and children (Egilman and Billings, 2005; Godwin and Jagatic, 1968; Teta et al., 1983; Robinson et al., 1979; McDonald et al., 1983; Newhouse and Thompson, 1965; McDonald et al., 1970; Greenburg and Lloyd Davies, 1974; Langer and McCaughey, 1982; Huncharek et al., 1989).

Friction products may contain up to 70% chrysotile asbestos. During the process of braking, asbestos fibers undergo abrasion and elevated temperatures which partially alters the structure of chrysotile and some fibers lose their fibrous character. This fiber conversion process is not complete and free asbestos fibers are found in brake wear dust. Lynch (1968) found up to 15% asbestos in wear dust after severe braking and Rohl et al. (1977) found from 0.5 to 15.1% chrysotile in brake wear debris. Free fibers longer than 5 urn have been found in brake wear debris (Rohl et al., 1976). Brake maintenance procedures can result in asbestos exposures to both mechanics and bystanders, particularly during the use of compressed air to blow debris from the brake assembly. In addition, extremely high concentrations of airborne asbestos fibers can be produced during the grinding of new brake linings if conducted without local exhaust ventilation (Rohl et al., 1976; Lorimer et al., 1976; Kauppinen and Korhonen, 1987). Exposures during sanding and grinding would be the same as seen in the asbestos friction product plants and would include high concentrations of fibers longer than 5 urn (Dement and Wallingford, 1990). Merewether and Price (1930) discussed significant exposures to asbestos during the manipulation of asbestos-containing brakes and clutches and stated that "the amount (of dust) is considerable at sawing and grinding machines" and recommended the use of local exhaust ventilation.

Numerous studies have demonstrated that mechanics who worked with asbestos containing brakes without dust-control measures were exposed to asbestos dust. This is particularly true when the mechanic grinds, files, or sands the new asbestos brake and uses compressed air or dry brushing to clean out wear dust from old asbestos brakes. Rohl et al. (1976) reported that chrysotile asbestos was found in all dust samples taken from car brake drums, with 2-15% in each sample in both fiber and fibril forms, with average concentrations from blowing the dust of 16 fibers/ml of air and that measurable concentrations were also found up to 75 feet from the actual worksite some 15 min after blowing out ceased. Lorimer et al. (1976) found mean fiber concentrations of 3.8 fibers/ml among New York brake repair workers, which is similar to other studies that show intact chrysotile fibers may be released from brake materials. Up to 8.2 f/cm3 was found during cleaning of the drum brakes of passenger cars using a compressed air jet to remove the brake dust Kauppinen and Korhonen (1987). Roberts et al. (1982) reported time-weighted average (TWA) exposures of about 0.2 f/cc and peak exposures of about 15 f/cc while using dry brushing, wet brushing, or compressed air during brake repair. Both the EPA and OSHA have issued guidance to reduce the risk of disease from asbestos exposure during brake work.

14. Asbestos Exposure and Mesothelioma

All forms of asbestos are carcinogenic to humans. There is sufficient evidence in humans and experimental animals for the carcinogenicity of all forms of asbestos (chrysotile, crocidolite, amosite, tremolite, actinolite, and anthophyllite). Asbestos causes mesothelioma and cancer of the lung, larynx, and ovary (IARC, 2012). The associations between exposure to asbestos, lung cancer, and mesothelioma have been well established in numerous epidemiological investigations. For talc that contains asbestiform fibers, previous Working Groups assessed studies on talc described as containing asbestiform tremolite and anthophyllite. These fibers fit the definition of asbestos. There is emerging epidemiological evidence that non-commercial amphibole fibers that are asbestiform have carcinogenic potential. These fibers are not technically “asbestos,” and they were never commercially marketed.

The conclusions reached in IARC Monograph 100 C (2012) about asbestos and its carcinogenic risks apply to chrysotile, actinolite, amosite, anthophyllite, crocidolite, tremolite fibers wherever they are found, and includes talc containing asbestiform fibers. All forms of asbestos including mineral substances such as talc or vermiculite that contain asbestos should also be regarded as carcinogenic to humans.

Malignant mesothelioma is a rare disease. It is reported in literature that 50 to 90% of individuals with pleural mesotheliomas have an identifiable history of asbestos exposure (Carbone et al., 2012a; Sebbag and Sugarbaker, 2001; Dodson and Hammer, 2011 pp 576; Strauchen, 2011) In an assessment of lung asbestos fiber burden and asbestos exposure history among patients diagnosed with pleural malignant mesothelioma, (Carbone et al., 2012b) 11 of 18 (61%) individuals reporting a negative history of asbestos exposure had lung fiber burden concentrations > 0.5 million fibers/dry gram of tissue. Similar results were reported by Leigh et al. (2002), revealing asbestos fibers in the lungs of 80% of Australian patients were not aware of having had asbestos exposure. These results suggest that people may not always be aware of the exposures they have suffered.

Individuals with known occupational exposures to asbestos cannot be recast into the “idiopathic” or “unknown exposure” category. When confronted with an individual who has a demonstrated mesothelioma and an occupational exposure to asbestos, the mainstream scientific community

recognizes that the cause of that mesothelioma is the asbestos exposure of the individual even if that exposure was "brief or low-level" (Welch, 2007). The consensus of the scientific community is that there is no demonstrable threshold of exposure to asbestos below which adverse health effects do not occur. Accordingly, "an occupational history of brief or low-level exposure should be considered sufficient for mesothelioma to be designated occupationally related" to asbestos exposure (Helsinki criteria, 2014). The World Health Organization (WHO, 1989) stated "The human evidence has not demonstrated that there is a threshold exposure level for lung cancer or mesothelioma, below which exposure to asbestos dust would not be free of hazard to health", and the International Programme for Chemical Safety (1998) reiterated this position. Asbestos-induced mesothelioma has a long latency period, usually 30 or more years, and the latency increases with lower levels of exposure (Browne, 1994; Bianchi et al., 2007). Unlike carcinoma associated with asbestos exposure, mesothelioma is not associated with cigarette smoking (Klaassen, 2013).

The scientific community is in consensus that even brief and low-level exposure to asbestos can cause mesothelioma. The mainstream scientific community has long recognized and continues to recognize today that there is no "safe" level of exposure to asbestos (World Trade Organization, 2000; Helsinki criteria, 2014). As noted by NIOSH, excessive cancer risks have been demonstrated at all fiber concentrations studied to date. Evaluation of all available human data provides no evidence for a threshold or for a "safe" level of asbestos exposure (NIOSH, 1980). These conclusions support what industry representatives were saying at the Selikoff conference in 1965, that the only safe level of exposure to asbestos to prevent disease is zero. It also supports the finding that nonmalignant respiratory diseases do not need to be present before cancer of the lung or mesothelioma can develop. At the New York Academy of Sciences conference on the Biological Effects of Asbestos in 1965, asbestos industry representatives from Britain and the U.S stated: "We do not believe there is a safe limit…Therefore, I would like it to be clearly understood that we do not accept 4 fibers/mL as a safe maximum limit in the asbestos industry" (Addingley, 1965), and, "Our own conclusions, as we began seeing what was happening in our own process, was that the only safe amount of asbestos dust exposure was zero and that the efforts in terms of achieving that lay basically in engineering, and, secondly, in education" (Wells, 1965).

There is inconsistency in the literature regarding linear dose response curves for asbestos exposure and malignant mesothelioma. It has been commonly reported that there is a dose-response relationship that is linear (risk increases with increased exposure) with no threshold (no safe level of exposure exists) (Lin et al., 2007; Dodson & Hammer, 2011). Other studies, primarily focusing on environmental asbestos and erionite mineral fiber exposures, have not reported a linear dose-response relationship between asbestos exposure and malignant mesothelioma (Carbone et al., 2012a; Carbone et al., 2012b), suggesting that some individuals may be more susceptible to asbestos induced malignant mesothelioma than others due to factors such as genetics, exposure to cofactors (ionizing radiation, Simian virus), and mineral fiber constituencies (Carbone et al., 2012b).

Complexity in defining the mechanisms of toxicity exits for malignant asbestos related diseases. Proposed mechanisms for the carcinogenicity of asbestos fibers as defined by the International Agency for Research on Cancer (IARC, 2012; EPA/IRIS, 2014b) include direct fiber-cell interaction with target cells and indirect interaction generated from cellular signaling pathways. The surface of asbestos fibers deposited in the lungs acquires iron that that cycles between the reduced and oxidized forms (Shannahan, 2011). This redox cycle may result in DNA lesions which may lead to apoptosis, gene mutations, chromosomal abberations, and cell transformation (Huang et al., 2011). Asbestos-induced reactive oxygen species (ROS) production may also result in p53 activation, and other cellular signals

including cytokines, chemokines and growth factors (Liu et al., 2013). The proposed events for asbestos carcinogenicity also include macrophage interaction, inflammasome activation associated with frustrated phagocytosis, release of cytokines and growth factors, and subsequent inflammation. Asbestos is considered to be both an initiator and a promotor of the carcinogenic process (Mossman et al., 2011).

A recent mechanism proposed for mesothelial cell transformation is that asbestos fibers induce necrotic cell death of human mesothelial cells, which results in the release of high-mobility group protein B1 (HMGB-1) in the extracellular space (Yang et al., 2006 and 2010). Secreted HMGB-1 induces initiates a chronic inflammatory response which includes an accumulation of macrophages and the release of inflammatory cytokines from macrophages, including TNF-α and IL-1β. "TNF-α activates the NF-β pathway, which increases the survival of human mesothelial cells after asbestos exposure, allowing cells with asbestos-induced DNA damage to divide rather than die, and if key genetic alterations accumulate, to eventually develop into malignant mesothelioma" (Carbone and Yang, 2012b).

Unfortunately, the rates of mesothelioma in the United States are not decreasing. The incidence of malignant mesothelioma in men has continued to rise in the past 50 years (Carbone et al., 2012). A recent Center for Disease Control (CDC) analysis of multiple-cause death records from 1999 – 2015 revealed that death rates from malignant mesothelioma increased from 2,479 deaths in 1999 to 2,597 deaths in 2015. This (CDC) study identified the trades of "pipelayers, plumbers, pipefitters, and steamfitter" as occupations with significantly elevated proportionate mortality ratios (PMR), reporting a PMR of 4.8 (3.7–6.1) (95% confidence interval) (Mazurek et al., 2017).

Iwatsubo et al (1998) found an excess of pleural mesothelioma in the lowest exposure group with an estimated total exposure between 0.001 and 0.49 f/cc-years. Twenty three percent of cases and 36% controls were exposed to less than 0.5 f/cc-years. The time-related pattern of exposure revealed a significantly elevated OR among workers whose exposure to asbestos was intermittent.

Rodelsperger et al. (2001) concluded there was a distinct dose-response relationship, even at low levels of exposure, with exposures from >0 to <0.5 f/cc-years showing a significantly increased risk of mesothelioma. Study results confirm the previously reported observation of a distinct dose-response relationship even at levels of cumulative exposure below 1 fiber year. Rodelsperger et al. (2001) stated: "In addition to asbestos exposure at the workplace, contact in the household and environmental exposure to asbestos are established causes of diffuse malignant mesothelioma."

Rolland et al. (2006) reported that a significant dose-response relationship was found between cumulative occupational exposure and pleural mesothelioma, even for the lowest category (greater than 0-0.07 f/cc-years, OR 2.8, 95% CI = 1.7-4.7).

Offermans et al. (2014) reported that for mesothelioma, hazard ratios (HRs) were significantly elevated in this study, even for the lowest tertile of CE median, 0.20 f-y/mL) based on FINJEM(HR = 2.69 [95%CI, 1.60 to 4.53]).

Lacourt et al (2014) reported that a clear dose-response relationship was observed between occupational asbestos exposure and pleural mesothelioma (OR=4.0 (99% CI 1.9 to 8.3) for men exposed at less than 0.1 f/mL-year vs 67.0 (99% CI 25.6 to 175.1) for men exposed at more than 10 f/mL-year). The study also suggests that the overall population attributable risk (Arp) in women is largely driven by non-occupational asbestos exposure arguing for the strong impact of such exposure in pleural mesothelioma occurrence.

Dodson and Hammer (2011) reported increased risk of mesothelioma at concentrations lower than 0.1 f/cc-years. At 0.1 f/cc-years, there was an excess of 7 cases/100,000 people exposed.

15. Early Knowledge of Libby Amphibole (LA) Asbestos Hazards

As of 1956, the State was aware of the presence of asbestos at the Zonolite mine and mill operation (MT State Report 8/13/1956). In a confidential report to Zonolite in 1956, the State warned of the toxicity of asbestos by stating that "the asbestos dust in the [vermiculite] dust in in the air is of considerable toxicity and is a factor in reducing the dustiness of the plant." The report expanded the asbestos toxicity discussion by describing the potential for respiratory fibrosis that is associated with asbestos exposure. "The asbestos fiber group…stimulate the formation of a diffuse fibrosis" (Report and Cover letter from Wake to Bleich, 9/21/56). In a report to Zonolite in 1959, the State further described the toxicity of asbestos by stating that, "According to Ellman (1933), Inhalation of asbestos dust must be expected sooner or later to produce pulmonary fibrosis, depending upon (a) length of exposure and (b) nature and concentration of the dust. Pulmonary asbestosis, once established, is a progressive disease with a bad prognosis; its treatment can only be symptomatic…." In this report, the State provided results of the 13 dust samples collected in the six floors of the dry mill. In addition to particle analysis to determine concentrations for comparison to the nuisance dust standard, the concentration of asbestos was also determined and reported (Report and Cover letter from Wake to Bleich, 1/12/1959). In a report to Zonolite in 1962, the State noted that dust concentrations had increased from the two previous evaluations (1956 and 1958 (reported in 1959)). The results of 20 dust samples were reported. Analyses were compared to exposure limits for mica, rather than nuisance dust, and asbestos. "The asbestos content of the dust is the controlling factor for making the maximum allowable concentration 5 mppcf." (Report and Cover letter from Wake to Bleich, 4/19/1962). In a report to Zonolite in 1963, the State reported that there was "as serious dust concentration, particularly in view of the quantity of asbestos known to be in the mixture" (Report and Cover letter from Wake to Bleich, 4/11/1963). The State sent additional communication to Zonolite in July of 1963, regarding the results of six vermiculite samples that were analyzed to determine the tremolite/actinolite content. The percentage of asbestos, reported as tremolite-actinolite ranged from 6.2 to 22.5 (Letter from Wake to Bleich, 7/3/1963). In a report to Zonolite in 1964, the State discussed the results of six dust samples conducted in the dry mill. In this report, the State specifically warned that asbestos exposure has the potential to cause cancer, including mesothelioma, citing work by Selikoff as well as British and South African investigators. The report further added that, "while the above situation does not apply specifically to the operations of your plant, the asbestos content of the material with which you are working appears to provide some serious potential for the development of disease if not properly controlled" (Report and Cover letter from Wake to Bleich, 5/11/1964).

In the early 1960s, asbestosis began appearing on the death reports of former vermiculite mine workers. Glenn Taylor, a worker at Zonolite was diagnosed with asbestosis in 1959 (Discharge Summary of Glenn Taylor, 3/20/1959). Glen Taylor died of asbestosis in 1961 (Death Certificate – Glenn Taylor, 9/15/1961). The State also had notice of deaths due to asbestos related pulmonary issues or corpulmonale (lung

related heart failure) of Zonolite employees: Charles Wagner, a mechanic, in 1961 (Death Certificate of Charles Michael Wagner, 10/14/1961); Albert Barney, a drymill worker in 1964 (Death Certificate of Albert Burr Barney, 1/12/1964), and; Walter McQueen, a miner, in 1966 (Death Certificate of Walter Leo McQueen, 10/30/1966).

16. Conclusion

Based on the information presented in this report, my reading of the case material pertaining to Grace Webster, my knowledge of the issues relating to Libby Amphibole Asbestos (LA) and asbestos in general, and my experience in the field of industrial hygiene, it is my opinion that Grace Webster was exposed the LA throughout her work involving Orton Company cones packed in vermiculite. Her work with vermiculite involved the disturbance of vermiculite contaminated with LA which dispersed asbestos fibers into the air and into her breathing zone, and this resulted in her mesothelioma.

Summary of Opinions

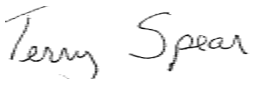
My opinions in this case are based on my education, training, and experience as an industrial hygienist, my knowledge, research, and review of industrial hygiene literature, testing and research that I have personally performed relating to asbestos, my review of case testimony, and case specific documents and manufacturers' materials. It is my opinion to a reasonable degree of scientific certainty that:

A. The facts stated in the report are sufficient to form a reliable basis for my opinion. I have reviewed the above mentioned materials in order to formulate my opinions in this case. The materials I have reviewed are typically relied upon by industrial hygienists to formulate opinions. Based on these materials, it is my opinion that Grace Webster was exposed to asbestos directly during the course of her work with Orton cones packed in vermiculite. Each exposure to asbestos in excess of background arising from her work with Orton cones packed in vermiculite without effective controls contributed to her overall cumulative asbestos dose. The exposures Grace Webster to visible dust containing asbestos fibers described by Karen Cahoon were many orders of magnitude greater than any generally accepted background levels and would be considered by any industrial hygienist to be dangerous exposures.

B. Based on my review, the peer-reviewed published industrial hygiene, epidemiology, and medical literature, as well as government studies, demonstrates within a reasonable degree of scientific certainty that friable asbestos-containing materials, and those that become friable through handling, pose as significant risk for causing malignant mesothelioma if effective measures to control dust exposures are not implemented. Mesothelioma does not result solely from exposure to background concentrations (0.00005 f/cc). The current literature establishes that mesothelioma results from direct or indirect occupational and non-occupational exposure (domestic and environmental) to asbestos above these background concentrations.

C. Karen Cahoon testified that visible dust was generated when Grace Webster retrieved cones packed in vermiculite from Orton boxes, when she dumped the boxes containing cones and vermiculite onto a tray, and during cleaning of the debris from the Orton cone boxes. Based on my review of Karen Cahoon's testimony, case materials and corporate documents, and research describing fiber release from source media containing less than 1% asbestos, Grace Webster's exposure to asbestos during the course of her work with Orton cones packed in vermiculite was many orders of magnitude greater background levels and contributed to her cumulative asbestos dose and substantially increased her risk of developing mesothelioma.

Terry Spear



References

Addison J., Davies L.S.T., Dwaneson A., Willey R.J. *The release of dispersed asbestos fibres from soils.* Historical Research Report. Research Report TM/88/14 (1988).

Addison, J. *Vermiculite: a review of the mineralogy and health effects of vermiculite exploitation.* Reg. Tox. Pharm. 21: 397-405 (1995).

Alexander BH, Raleigh KK, Johnson J, Mandel JH, Adgate JL, Ramacandran G, Messing RB, Eshenaur T, Williams A. 2012. Radigraphic evidence of nonoccupational asbestos exposure from processing Libby vermiculite in Minneapolis, Minnesota. Env Health Pers 120(1): 44-49.

Amandus HE, Wheeler PE, Jankovic J, Tucker J. 1987. The morbidity and mortality of vermilite miners and millers exposed to tremolite-actinolite: Part I. Exposure estimates. Am J of Ind Med; 11: 1-14.

Amandus HE, Wheeler R. 1987. The morbidity and mortality of vermilite miners and millers exposed to tremolite-actinolite: Part II. Mortality. Am J of Ind Med; 1: 15-26.

Antao VC, Larson TC, Horton DK. 2012. Libby vermiculite exposure and risk of developing asbestos-related lung and pleural diseases. Curr Opin Pulm Med. 18: 161-167.

Agency for Toxic Substances and Disease Registry (ATSDR 2003a). U.S. Department of Health and Human Services, Agency for Toxic Substances and Disease Registry, ATSDR, Public Health Assessment, Libby Asbestos Site Libby, Lincoln County, Montana, EPA Facility ID: MT0009083840 (May 15, 2003).

Atkinson, GR, Rose D, Thomas K, Jones D, Chatfield EJ, Going JE. 1982. Collection, analysis and characterization of vermiculite samples for fiber content and asbestos contamination. Task 32, EPA Prime Contract No. 68-01-5915.

Bartrip PWJ. 2003. Review: History of asbestos related disease. Postgrad Med. J. 80: 72-76.

Bandli BR, Gunter ME, Twamley B, Foit FF, Jr., and Cornelius, SB. 2003. Optical, compositional, morphological, and X-ray data on eleven particles of amphibole from Libby, Montana, U.S.A. Canadian Mineralogist, 41, 1241-1253.

Bassett WA. 1959. The origin of the vermiculite deposit at Libby, Montana. Am. Mineral. 44: 282-299.

Boettcher AL. 1966. Vermiculite, hydrobiotite, and biotite in the rainy creek igneous complex near Libby, Montana. Clay Minerals. 6: 283-296.

Browne K. Asbestos-related disorders. In: Parkes WR, editor. Occupational Lung Disorder. Oxford: Butterworth-Heinemann; 1994. pp. 411–504.

Cartier P. 1952. Abstract of Discussion. Arch Indust Hyg Occup Med. 5: 262-263.

Case B. 2006. Mesothelioma update for Libby, Montana: Occupational and non-occupational. Lung Cancer 54 (S1) S10.

Castleman, B. 1996. Asbestos: Medical and Legal Aspects, Fourth Edition. Aspen Law and Business. 270 Sylvan Avenue, Englewood Cliffs, NJ 07632.

CDM Smith (CDM,2009). *Former Export Plant Site Final Remedial Investigation Report, Operable Unit 1, Libby Asbestos Site, Libby, MT* (2009).

Colorado Department of Public Health and Environmental, Hazardous Materials and Waste Management Division. Retrieved from http://www.cdphe.state.co.us/hm/asbestos/111021riskppt.pdf

Cooke, W.E. 1924. Fibrosis of the Lungs Due to the Inhalation of Asbestos Dust. Brit. Med. J. 2:147.

Cooke WE, 1927. Pulmonary Asbestosis. Brit. Med. J. 2: 1024-1025.

Cowen BW. 1997. Elevated Asbestos Exposures from a Building Demolition Which Contained Vermiculite Insulation. Presentation at the American Industrial Hygiene Association Conference and Exposition (AIHCe, 1997). Abstract available at www. AIHA.org.

Deane L. 1898. Report on the health of workers in asbestos and other dusty trades, in HM Chief Inspector of Factories and Workshops, 1899, Annual Report for 1898, pp. 171–172, HMSO London (see also the Annual Reports for 1899 and 1900, p502) as reported in Gee and Greenberg, 2002.

Doll, R. *Mortality from Lung Cancer in Asbestos Workers*. Brit. J. Indust. Med. 12: 81-86 (1955).

Dressen, W.C. et al. *Study of Asbestosis In The Asbestos Textile Industry.* U.S. Treasury Department Public Health Service. Public Health Bulletin. No 241 (August 1938).

Drinker, P. & Hatch, T. Industrial Dust, Hygienic Significance, Measurement and Control (1936).

Egbert DS, Geiger AJ. 1936. Pulmonary Asbestosis and Carcinoma. Amer. Rev. Tuberc. 34: 143-150.

Ewing, W.M., Hays, S.M., Hatfield, R., Longo, W.E., & Millette, J.R. (2010). Zonolite attic insulation exposure studies. International Journal of Occupational and Environmental Health,16(3), 279–290.

Gloyne SR. 1933. The Morbid Anatomy and Histology of Asbestosis. Tubercle. 14: 440-558.

Gloyne SR. 1935. Two Cases of Squamous Carcinoma of the Lung Occurring in Asbestosis. Tubercle. 1: 4-10.

Gloyne SR. 1936. A Case of Oat Cell Carcinoma of the Lung Occurring in Asbestosis. Tubercle. 18: 100-101.

Greenberg M. 1999. A Study of Lung Cancer Mortality in Asbestos Workers. Am Jour of Ind Med. 3: 331-347.

Gunter ME, Brown BM, Bandli BR, and Dyar MD. 2003. Composition, Fe3+/∑Fe, and crystal structure of non-asbestiform and asbestiform amphiboles from Libby, Montana, U.S.A. Am Mineral. 88: 1970-1978.

Gunter ME and Sanchez MS. 2009. Amphibole forensics: Using the composition of amphiboles to determine their source, the Libby, Montana, example. Amer Minerologist. 94: 837-840.

Helsinki criteria, 2014. International Conference on Monitoring and Surveillance of Asbestos Related Diseases 11-13 February 2014, Finland. Finnish Institute of Occupational Health.

Hoffman, F.L. Mortality from Respiratory Diseases in Dusty Trades (Inorganic Dusts). 1918. Bulletin of the United States Bureau of Labor Statistics. Industrial Accidents and Hygiene Serious. No 17.

Holleb HB, Angrist A. 1942. Bronchiogenic Carcinoma in Association with Pulmonary Asbestosis. Amer. J. Path. 18: 123-131.

Homburger F. 1943. The Co-incidence of Primary Carcinoma of the Lungs and Pulmonary Asbestosis. Amer. J. Path. 19 (5): 797 -807.

Hueper WC. 1942. Occupational tumors and allied diseases. Springfield, Ill. Charles C. Thomas. 399-405.

Journal of the American Medical Association (JAMA). November 25, 1944. Editorial: Environmental Cancer. 836.

Klaassen CD. 2013. Casarett & Doull's Toxicology, the Basic Science of Poisons. 8th Edition. ISBN: 9780071769235.

Kriegel WW. 1940. Summary of occurrence, properties, and uses of vermiculite at Libby, Montana. Bulletin of The Amer Ceramic Soc. 19 (3): 94-97.

Larson TC, Antao VC, Bove FJ. 2010. Vermiculite worker mortality: estimated effects of occupational exposure to Libby amphibole. J Occup Environ Med. 52(5): 555-560.

Lin, R-T, Takahashi, K, Karjalainen, A et al. Ecological association between asbestos related diseases and historical asbestos consumption: an international analysis. *Lancet*. 2007; **369**: 844–849

Lockey JE, Jarabek, A, Carson A, McKay R, Harber P, Khoury P, Morrison J, Wiot J, Spitz H, Brooks S. 1983. Health issues related to metal and non-metallic mining. Pulmonary Hazards from Vermiculite Exposure. Boston: Butterworth. 303-315.

Lockey JE, Brooks SM, Jarabek M, Khoury PR, McKay RT, Garson A, Morrison JA, Wiot JF, Spitz HB. 1984. Pulmonary changes after exposure to vermiculite contaminated with fibrous tremolite. Am Rev Respir Dis. 129(6): 952-958.

Lynch, K.M. & Smith, W.A. *Asbestosis Bodies in Sputum and Lung.* Asbestosis. Vol 95, No 9 (1935).

Lynch KM, Smith WA. 1939. Pulmonary Asbestosis V. A Report of Bronchial Carcinoma and Epithelial Metaplasia. Amer. J. Cancer. 36: 567-574.

McDonald JC, McDonald AD, Armstrong B, Sebastien P. 1986. Cohort study of mortality of vermiculite miners exposed to tremolite. Brit J Ind Med. 43: 436-444.

McDonald JC, Harris J, Armstrong B. 2004. Mortality in a cohort of vermiculite miners exposed to fibrous amphibole in Libby, Montana. Occup Environ Med. 2004 61:363-366.

McKean, D. Toxicity and Risk Assessment, U.S. Environmental Protection Agency (EPA), EPA, Region 8 (2011).

Meeker GP, Bern AM, Brownfield IK, Lowers HA, Sutley ST, Hoefen TM, Vance JS. 2003. The Composition and Morphology of Amphiboles form the Rainy Creek Complex , Near Libby, Montana. American Minerologist; 88:1955-1969.

Merewether, E.R.A. *The Occurrence Of Pulmonary Fibrosis And Other Pulmonary Affections In Asbestos Workers*. Journal of Industrial Hygiene. Vol 12, No 6 (1930a).

Merewether, E.R.A. & Price, C.W. *Report on Effects of Asbestos Dust on the Lungs and Dust Suppression in the Asbestos Industry.* Processes giving Rise to Dust and Methods for its Suppression (1930b).

Merewether, E.R.A. *A Memorandum on Asbestosis.* Tuberlce (November 1933).

Moolgavkar SH, Turm J, Alexander DD, Lau EC, Cushing CA. 2010. Potency factors for risk assessment at Libby, Montana. Risk Analysis. 30(8): 1240-1248.

Moatamed F, Lockey JE, Parry WT. 1986. Fiber contamination of vermiculites: a potential occupational and environmental health hazard. Envir Research. 41: 207-218.

National Institute for Occupational Safety and Health. Workplace Exposure to Asbestos: Review and Recommendations: NIOSH/OSHA Asbestos Work Group Recommendations. Department of Health and Human Services, 1980: 81-103.

National Institute for Occupational Safety and Health (NIOSH) Division of Respiratory Disease Studies. 2008. Work-Related Lung Disease Surveillance Report 2007. page 181: Malignant mesothelioma: Counties with highest age-adjusted death rates (per million population), U.S. residents age 15 and over, 2000–2004. DHHS/ CDC/ NIOSH.

Noonan CW, Pfau JC, Larson TC, Spence MR. 2006. Nestled case-control study of autoimmune disease in an asbestos – exposed population. Envir Health Pers. 114(8): 1243-1247.

Oliver, T. 1927. Clinical Aspects of Pulmonary Asbestosis. Brit. Med. J. 2: 1026-1027.

Oliver, T. 1927. Pulmonary Asbestosis in its Clinical Aspects. J. Ind. Hyg. 9: 483-485.

Pancoast HK, Miller TG, Landis HM. 1918. A Roentgenologic Study of the Effects of Dust Inhalation upon the Lungs. Republished. American Journal of Roent. 5: 129-138.

Pardee JT, Larsen ES. 1929. Deposits of vermiculite and other minerals in the Rainy Creek District, near Libby, Montana: USGS Bulletin; 805: 17-28.

Peipins LA, Lewin M, Campolucci S, Lybarger JA, Miller A, Middleton D, Weis C, Spence M, Black B, Kapil V. 2003. Radiographic abnormalities and exposure to asbestos-contaminated vermiculite in the community of Libby, Montana, USA. Environ Health Perspect; 111(14):1753-1759.

Potter MJ. 1997. Vermiculite. U.S. Geological Survey- Minerals Information. Retrieved June 25, 2013 from: http://minerals.usgs.gov/minerals/pubs/commodity/vermiculite/710497.pdf.

Rohs, AM, Lockey JE, Dunning KK, Shukla R, Fan H, Hilbert T, Borton E, Wiot J, Meyer C, Shipley RT, LeMasters GK, Kapil V. 2008. Low-level fiber-induced radiographic changes caused by Libby vermiculite. 177: 630-637.

Sanchez, MS, Gunter, ME, and Dyar, MD. 2008. Characterization of historical amphibole samples from the former vermiculite mine near Libby, Montana, USA. European Journal of Mineralogy, 20, 1043-1053.

Selikoff IJ, Churg J, Hammond EC. 1963. Relation Between Exposure to Asbestos and Mesothelioma. N Eng J Med. 272: 560-565.

Selikoff IJ, Churg J, Hammond EC. 1964. Asbestos Exposure and Neoplasia. Jour Amer Med Assoc. 188: 142-146.

Selikoff IJ, Churg J, Hammond EC. 1965. The Occurrence of Asbestosis Among Insulation Workers in the United States. Am N Y Acad Sci 1965-66. 132: 139-155.

Spear TM, Hart JF, Spear TE, Loushin MM, Shaw N, Elashheb MI. 2012. The presence of asbestos-contaminated vermiculite attic insulation and/or other asbestos containing materials in homes and the potential for living space contamination. Journal of Environmental Health, 75:3, 24-29.

Sullivan PA. 2007. Vermiculite Respiratory Disease and Asbestos Exposure in Libby, Montana: Update of a Cohort Mortality Study. Environ Health Perspect. 115 (4) 579 – 585.

United States Environmental Protection Agency (USEPA) 2000. Sampling and analysis of consumer garden products that contain vermiculite. EPA 744-R-00-010.

United States Environmental Protection Agency (EPA 2003a). U.S. Environmental Protection Agency, Final Draft, Pilot Study To Estimate Asbestos Exposure from Vermiculite Attic Insulation, Research Conducted in 2001-2002. Prepared for: Fibers and Organic Branch, National Program Chemical Division, Office of Pollution Prevention and Toxics, U.S. Environmental Protection Agency. Prepared by Versar Inc (May 21, 2003).

United States Environmental Protection Agency (EPA 2003b). U.S. Environmental Protection Agency, Libby Asbestos Site Residential/Commercial Cleanup Action Level and Clearance Criteria, Technical Memorandum Draft Final (December 15, 2003).

United States Environmental Protection Agency (EPA 2007a). U.S. Environmental Protection Agency, Summary Report for Data Collected Under the Supplemental Remedial Investigation Quality Assurance Project Plan (SQAPP) for Libby, Montana (October 23, 2007).

United States Environmental Protection Agency (USEPA) 2009. EPA announces public health emergency in Libby, Montana. EPA to move aggressively on cleanup and HHS to assist area residents with medical care. Retrieved January 12, 2013 from: http://yosemite.epa.gov/opa/admpress.nsf/6427a6b7538955c585257359003f0230/0d16234d252c98f9852575d8005e63ac!OpenDocument.

United States Environmental Protection Agency (USEPA) 2012a. Region 8- Libby asbestos, background on the Libby asbestos site. Retrieved January 12, 2013 from: http://www.epa.gov/libby/background.html.

United States Environmental Protection Agency (EPA) 2014a. Toxicological Review of Libby Amphibole Asbestos: In Support of Summary Information on the Integrated Risk Information System (IRIS). EPA 636-R-11-002F.

United States Environmental Protection Agency (EPA) 2014b. Libby Asbestos Superfund Site Residential and Commercial Properties Operable Unit 4 Libby, Montana Remedial Investigation Report Contract No. W9128F-11-D-0023 Task Order No.: 0003 and 0007 June 2014.

Vinikoor LC, Larson TC, Bateson TF, Birnbaum L. 2012. Exposure to asbestos-containing vermiculite ore and respiratory symptoms among individuals who were children while the mine was active in Libby, Montana. Env Health Pers. 118(7): 103-1038.

Vorwald, A.J., Durkan, T.M. & Pratt, P.C. *Experimental Studies of Asbestosis.* AMA Archives of Industrial Hygiene and Occupational Medicine. Vol 3, No 1 (January 1951).

Wagner, J.C., Sleggs, C.A. & Marchand, P. *Diffuse Pleural Mesothelioma and Asbestos Exposure in the North Western Cape Province*. British Journal of Industrial Medicine (1960).

Wedler HW. 1943. Lung Cancer in Asbestos Patients. Deut Arch Klin Med. 191: 189-209.

Welch LS. Asbestos exposure causes mesothelioma, but not this asbestos exposure: an amicus brief to the Michigan Supreme Court. Int J Occup Environ Health. 2007 Jul-Sep;13(3):318-27.

Whitehouse AC, Black CB, Heppe MS, Ruckdeschel J, Leven SM. 2008. Environmental Exposure to Libby Asbestos and Mesotheliomas, American Journal of Industrial Medicine, D01 10.1002/ajim.20620.

Wyers, H. *Asbestosis.* Postgraduate Medical Journal (December 1949).

Attachment 1

Curriculum Vitae

December 2015

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EDUCATION

Baccalaureate Degree (B.A.): **Microbiology**;June 1975; University of Montana**,** Missoula, Montana.

Master of Science (M.S.) Environmental Health; December 1980, University of Minnesota.

Doctor of Philosophy (Ph.D.): **Environmental Health**; April 1996, University of Minnesota
Dissertation Title: Assessment of Workers' Exposure to Lead-Containing Aerosol

PROFESSIONAL EXPERIENCE

Employment (full-time):

Faculty; Safety, Health and Industrial Hygiene Department: 8/1983 to 5/2012
Montana Tech of the University of Montana.
Assistant Professor (1983-1988); Associate Professor (1989-96); Full Professor, Tenured (1997- 2012); Department Head (2002 - 2012); Professor Emeritus (2012 - present).
Developed and taught academic courses in safety and industrial hygiene in the Montana Tech campus and distance learning programs. Involved in all aspects of advising, developing, and directing of student research and academic progress.

Senior Engineer, 11/1980 to 8/1983
EG&G Idaho Inc., Idaho National Engineering Laboratory (INEL), Idaho Falls, Idaho. Provided industrial consultation to facility management, including investigation and monitoring of work environments, ventilation evaluations, design and work practice reviews, hazardous materials oversight, and worker education and training.

Related Professional Experience:

Affiliate Professor: 9/1981 - 5/1982
University of Idaho, Idaho Falls, Idaho. Instructor for Mines 433,
Environmental Health I, Industrial Hygiene, and Mines 434, Environmental
Health II, Occupational Stress.

Consulting: 1985 - Present
Provide consultation to a variety of general industry and mining companies on program document development, health and safety compliance auditing, regulatory issues, industrial hygiene field sampling, on-site hazard assessments, and training. Over 25 years experience in providing expert witness testimony involving consultation and participation in personal injury and illness liability litigation cases for plaintiffs, defendants, private industry, and insurance companies.

RESEARCH AWARDS

Weatherization protocols in homes containing vermiculite and other asbestos-containing material. U.S. Department of Health and Human Services REACH Grant, December 2006.

Yellowstone Winter Use Personal Air Monitoring, Rocky Mountains Cooperative Ecosystem Studies Unit, Cooperative Agreement Number H1200040001, January 2006. Yellowstone Winter Use Air Monitoring, continuation of UMT 15, *J1580050167,* 2006.

Yellowstone Winter Use Personal Air Monitoring, Rocky Mountains Cooperative Ecosystem Studies Unit, Cooperative Agreement Number H1200040001, January 2005.

Occupational Exposure to Submicron Particle Mass and Number Concentrations from Diesel Emissions In an Underground Mine, Supplemental Research Training Grant, NIOSH, 2002 - 2003.

Lead Exposure Associated with Weatherization Activities in Homes Containing Lead Based Paint, Department of Energy, Spring 2002.

Chemical speciation of two different sources of copper slag, Supplemental Research Training Grant, NIOSH, 2001 - 2002.

Airborne assessment of abrasive blasting aerosol using copper slag abrasive, Supplemental Research Training Grant, NIOSH, 1999 - 2000.

Assessment of workers' exposure to lead containing aerosols, International Lead and Zinc Research Organization (LZRO), 1994 -1996.

Assessment of workers' exposures to lead-containing aerosols during smelter remediation, Minerals Research Institute, 1993 - 1994.

Fugitive dust emissions and worker exposure during furnace slag processing, Minerals Research Institute, 1991.

Volatile organics released from water sprays, Minerals Research Institute, 1989 - 1990.

PEER-REVIEWED PUBLICATIONS

Dale J. Stephenson, Ashley Kunz, Julie F. Hart, Terry M. Spear, Emily J. Zamzow. Snowmobile Noise Exposure Monitoring of Yellowstone National Park Employees. *Intermountain Journal of Sciences*, Vol. 20, No. 1-3, September 2014.

Julie F. Hart , Terry M. Spear, Mohamed Elashheb, Kristopher D. Hutchings, Richard R. Rossi. Evaluating the impact of weatherization measures in homes that contain vermiculite insulation and/or other asbestos containing materials. To be submitted for publication in 2015.

Spear, TM, Julie F. Hart, JF, Spear, TE, Loushin, M, Shaw, N, Elashheb, MI. The presence of asbestos-contaminated vermiculite attic insulation and/or other asbestos containing materials in homes and the potential for living space contamination. *Journal of Environmental Health,* Volume 75. Number 3, 2012.

Tony J. Ward, Terry M. Spear, Julie F. Hart, James S. Webber & Mohamed I. Elashheb. Asbestos in Tree Bark A Review of Findings for this Inhalational Exposure Source in Libby, Montana. *Journal of Occupational & Environmental Hygiene.* 9: 387–397, April, 2012. ISSN: 1545-9624 print / 1545-9632 online.

Mohamed I. Elashheb, Terry M. Spear, Julie F. Hart, James S. Webber, and Tony J. Ward. "Libby Amphibole Contamination in Tree Bark Surrounding Historical Vermiculite Processing Facilities". *Journal of Environmental Protection*. Vol.2, No. 8, October 2011) . DOI: 10.4236/jep.2011.28122.

V. Balasubramanian, T.M. Spear, J.F. Hart, J.D. Larson: Evaluation of Surface Lead Migration in Pre-1950 Homes: An On-Site Hand-Held X-Ray Florescence Spectroscopy Study. *Journal of Environmental Health*. Volume 23, Number 10, pp. 14-19, 2011.

Julie F. Hart, Tony J. Ward, Terry M. Spear, Richard J. Rossi, Nicholas N. Holland, and Brodie G. Loushin. "Evaluating the Effectiveness of a Commercial Portable Air Purifier in Homes with Wood Burning Stoves: A Preliminary Study". *Journal of Environmental and Public Health*, Volume (2011), Article ID 324809. Doi:10.1155/2011/324809.

Julie F. Hart, J.F., Spear, T.M., Ward, T.J., Baldwin, C.E., Salo, M.N. and Elashheb, M.I. "An evaluation of potential occupational exposure to asbestiform amphiboles near a former vermiculite mine", *Journal of Environmental and Public Health,* vol. 2009, Article ID 189509. Doi:10.1155/2009/189509.

Ward, T.J., Hart, J.F., Spear, T.M., Meyer, B.J., and Webber, J.S. , "Fate of Libby Amphibole Fibers When Burning Contaminated Firewood" , *Environ. Sci. Technol.* 2009, 43, 2878-2883.

Hart, J.F., Ward T.J., Spear T.M., Crispen, K., Zolnikov, T.R. "Evaluation of asbestos exposures during firewood harvesting simulations in Libby, Montana - Preliminary Data," *Ann. Occup. Hyg.*Volume 51, Number 8, November 2007.

PEER-REVIEWED PUBLICATIONS (continued)

Spear, T.M., Hart, J., Stephenson, D.J., Yellowstone Winter Use Personal Exposure Monitoring, Rocky Mountains Cooperative Ecosystem Studies Unit (RM-CESU), RM-CESU Cooperative Agreement Number:J1580050167**,** June 1, 2006.

Ward, T.J., Spear, T.M., Hart, J.; Noonan, C., Holian,A., Getman, M., Webber, J.S. "Trees as reservoirs for amphibole fibers in Libby, Montana", *Science of The Total Environment*, Vol. 367, Issue 1, August 2006.

Stephenson, D.J., Spear, T.M., Lutte, M.G. "Evaluation of a Direct Reading Sampling Method to Measure Exposure to Diesel Particulate Matter in an Underground Metal Mine," *Mining Engineering*, Vol. 58, No. 8, August 2006.

Spear, T.M., Stephenson, D.J., Yellowstone Winter Use Personal Exposure Monitoring, Rocky Mountains Cooperative Ecosystem Studies Unit (RM-CESU), RM-CESU Cooperative Agreement Number: H1200040001, June 1, 2005.

Wilson, T.B., . Douglass, R.J., Spear, T.M., Hart, J.F., and Norman, J.B. "Evaluation of protective clothing for handling small mammals potentially infected with aerosol-borne zoonotic agents," *Intermountain Journal of Sciences*, Vol. 8(1), 2002.

Spear, T.M., Stephenson, D., Seymour, M., "Characterization of aerosol generated during abrasive blasting with copper slag," Presented atInhaled Particles IX, Robinson College, Cambridge, UK and published in *Annals of Occupational Hygiene*, Vol. 46, Supplement, pp. 296-299, 2002.

Stephenson, D., Spear, T.M., Seymour, M, and Cashell, L., "Airborne Exposure to Heavy Metals and Total Particulate During Abrasive Blasting Using Copper Slag Abrasive," *Appl. Occup. Environ. Hyg.,* Volume 17(6): 437-443, 2002.

Spear, T.M., Cannell, C.E., "Mixmaster exposure to dust during mixing of wildland fire retardant chemicals," *International Journal of Wildland Fire*, Volume 11(1), pp. 65-73, 2002.

Spear, T.M., DuMond, J.W., Lloyd, C.J. and Vincent, J.H., "An Effective Protection Factor Study of Primary Lead Smelter Workers," *Appl. Occup. Environ. Hyg.,* Volume 15(2): 235-244 (2000).

Spear, T.M., Hardgrove, R., Norman, J.B., Wulf, D.T., and Rossi, R.J. "The Effects of Strapped Spectacles on the Fit Factors of Three Manufactured Brands of Full Facepiece Negative Pressure Respirators," *Ann. Occup. Hyg.*, Vol. 43 (3): 193-199 (1999).

Spear, T.M., Svee, W., Vincent, J.H., Stanisich, N. "Chemical Speciation of Lead Dust Associated With Primary Lead Smelting," *Environmental Health Perspectives*. Vol. 106(9): 565-571 (1998).

Spear, T.M., Werner, M.A., Bootland, J.H., Murray, E.P., Gurumurthy, R. and Vincent, J.H. "Assessment of Particle Size Distributions of Health-Relevant Exposures of Primary Lead Smelter Workers," *Ann Occup. Hyg.*, Vol. 42, No. 2, pp. 73-80 (1998).

PEER-REVIEWED PUBLICATIONS (continued)

Vincent, J.H., Brosseau, L.M., Ramachandran, G., Tsai, P.J., Spear, T.M., Werner, M.A., McCullough, N.V. "Current issues in exposure assessment for workplace aerosols." In: Inhaled Particles VII (ed. Ogden TL), *Ann Occup. Hyg.*, *41 (Suppl. 1)*, 607-614 (1997).

Spear, T.M., Werner, M.A., Bootland, J., Harbour, A., Murray, E.P., Rossi, R. and Vincent, J.H., "Comparison of Sampling Methods for Personal Sampling of Inhalable and 'Total' Lead and Cadmium Containing-Aerosols in a Primary Lead Smelter," *Am. Ind. Hyg. Ass. J.*, 58:893-899 (1997).

Vincent, J.H., Werner, M.A., Tsai, P.-J. And Spear, T.M. (1996). "Studies of occupational aerosol exposures and the impact of introducing new criteria for standards." In: *Occupational Hygiene Solutions* (ed. G.S. Hewson), proceedings of the 15th Annual Conference of the Australian Institute of Occupational Hygienists, Tullamarine, Victoria, Australia, pp 33-41.

M.A. Werner, T.M. Spear and J.H. Vincent. "Investigation Into The Impact of Introducing Workplace Aerosol Standards Based On The Inhalable Fraction." Presented at the Second International Symposium on Modern Principles of Air Monitoring (Sälen, Sweden, February, 1996) and published in *The Analyst*, Vol 21 (1207-14), 1996.

PROFESSIONAL PRESENTATIONS

"Evaluating the Impact of weatherization measures in homes that contain vermiculite insulation and/or other asbestos containing materials". Northwest Occupational Health Conference, Pasco, WA. October 2011.

"Weatherization of houses containing vermiculite or other ACM". Spear, T.M. and Hart, J.F., ASTM Johnson Conference on Asbestos. July 25-29, 2011.

"The Abundance of Libby Amphibole in Vermiculite Insulation in Homes". Spear, T.M. Pacific Northwest International Section (PNWIS) of the Air & Waste Management Association (A&WMA). Nov 3-5, 2010.

"Evaluating potential asbestos pathways from vermiculite attic insulation and asbestos containing materials in Western Montana homes- Baseline Results". Spear, T.M. Northwest Occupational Health Conference, Vancouver, B.C., October 2009.

"Evaluation of Asbestos Exposures during Firewood Harvesting Simulations in Libby, Montana". Ward, T. J., Spear, T., and Hart, J. 2008 ASTM Johnson Conference, Burlington, VT, July 15, 2008 (poster).

"Trees as Reservoirs for Amphibole Fibers in Libby, Montana". Presented at the Mine Design, Operations & Closure Conference, April 2007.

"Winter use air monitoring in Yellowstone National Park". Presented at the Pacific Northwest American Industrial Hygiene Conference, October 2006.

"Occupational exposure to Snowmobile emissions in Yellowstone National Park," Presented at the American Industrial Hygiene Conference and Exposition, May 2005.

PROFESSIONAL PRESENTATIONS (continued)

"Occupational Exposure to Submicron Particle Mass and Number Concentrations from Diesel Emissions In an Underground Mine," Presented at the American Industrial Hygiene Conference and Exposition, May 2003.

"Demonstrating an association between exposure and risk of disease," Montana Trial Lawyers Association Scientific Evidence Seminar, April 25, 2003.

"Worker exposure to dust and heavy metals during abrasive blasting using copper slag." Presented at the Pacific Northwest American Industrial Hygiene Conference, October 2001.

"Characterization of aerosol generated during abrasive blasting with copper slag." Presented at the Inhaled Particles IX Symposium in Cambridge, UK, September 2001.

"Comparison of Methods for Personal Sampling of Inhalable and Total Abrasive Blasting Aerosol." Presented at the American Industrial Hygiene Conference and Exposition, May 2001.

"Airborne Exposure Assessment of Abrasive Blasting Aerosol Using Copper Slag Abrasive." Presented at the American Industrial Hygiene Conference and Exposition, May 2000.

"Respirator protection factors." Presented at the 19th Annual Lead Occupational Health Conference, Chicago, IL. October, 1997.

"An effective protection factor study at a primary lead smelter." Presented at and published in the abstracts of the American Industrial Hygiene Conference and Exposition, May 1996.

"The impact of introducing workplace standards based on the inhalable fraction." Presented at the Montana Safety and Health Conference, April 1996.

"Characterization of lead aerosol size distribution in a primary lead smelter." Presented at the Montana Academy of Sciences, April 1994.

"Inhalable Fungal Aerosol Exposure in a Wood Pellet Plant." Presented at the American Industrial Hygiene Conference and Exposition, May, 1993.

HONORS

Professor Emeritus, June 2012, Montana Tech of the University of Montana
Distinguished Industrial Hygienist Award, 2012, Pacific Northwest Section - American Industrial Hygiene Association.
NIOSH Fellowship, 1994 -1995, University of Minnesota
NIOSH Scholarship, 1979, University of Minnesota
NIOSH Scholarship, 1978, Montana Tech.

ADMINISTRATIVE EXPERIENCE

Program Manager: M.S. Distance Learning/Professional Track Industrial Hygiene Program
Head (2002-2012): Safety, Health & Industrial Hygiene Department, Montana Tech of The University of Montana
Director (2002-2012): Industrial Hygiene Graduate Program, Montana Tech of the University of Montana
Planning Committee: Big Sky Section American Society of Safety Engineers
AIHA and ASSE Student Section: Faculty Representative

Principal Investigator, Eleven funded research grants
Coordinate and direct graduate student research

PROFESSIONAL/COMMUNITY/UNIVERSITY ACTIVITIES

Technical Advisor to the Libby Area Technical Advisory Committee. Jan.2009 to Jan, 2012.
Past Member: Expert panel on "Potential Environmental Impacts of Dust Suppressants: Avoiding Another Times Beach". Sponsored by U.S. Environmental Protection Agency and the University of Nevada Las Vegas.
Past Member: Libby Amphibole Research Symposium, Libby, MT.
Past Faculty Representative, AIHA and ASSE Student Sections.

Past Member, Curriculum Review Committee, Montana Tech of The University of Montana
Past Member, Chancellor's Advisory Committee, Montana Tech of The University of Montana
Past Member, Graduate Council

PROFESSIONAL SOCIETIES

American Industrial Hygiene Association
Americas Section, International Society for Respiratory Protection